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A SYSTEM OF MEDICINE.

A

SYSTEM OF MEDICINE.

EDITED BY

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VOLUME THE SECOND,

CONTAINING

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LOCAL DISEASES.

§ I.—DISEASES OF THE NERVOUS SYSTEM.

A. GENERAL NERVOUS DISEASES.

B. PARTIAL NERVOUS DISEASES.

1. DISEASES OF THE HEAD.
2. DISEASES OF THE SPINAL COLUMN.
3. DISEASES OF THE NERVES.

§ II.—DISEASES OF THE DIGESTIVE SYSTEM.

A. DISEASES OF THE STOMACH.

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§ I.—*DISEASES OF THE NERVOUS SYSTEM.*

INTRODUCTION.

BY THE EDITOR.

A FEW words are necessary to explain the principle of classification adopted with regard to the diseases which find their place in this, the first, section of "partial diseases," or "affections of particular organs."

The maladies which are treated of in these volumes have been in the first place divided into two large groups, "general" and "local," and the reason for such division has been assigned (Classification of Diseases, vol. i. p. 24). Some diseases are "general,"—that is to say, they appear so to affect the whole body at once, that all its functions are impaired or altered; and not only so, but they are implicated to such an equality of degree that it is always difficult, and sometimes impossible, to say upon which system of organs, if upon any, the greater weight of the burden falls. Of such diseases the *first* volume of this System of Medicine contained the history. In the present, the *second* volume, we have to deal with another class of affections, the "local" or "partial," viz. with those in which we have little or no difficulty in localising the disease; maladies with regard to which we say at once that they are diseases of the nervous system, or of one portion of the digestive system. It is not intended that such diseases are accurately limited to the particular systems from which they derive their names; for we know well that the digestion is, or may be, disturbed in epilepsy, in apoplexy, and in paraplegia; and, on the other hand, that no severe perturbation can occur in the stomach without the simultaneous development of some corresponding disturbance in the nervous centres. Furthermore, we know that there is sometimes great difficulty in determining whether we have to deal with a disease universal in its distribution at its onset, or with some primary lesion of a particular organ, the secondary effects of which are general; or, even allowing that some particular organ is especially affected, the affection may be of such kind that it is sometimes a matter of grave doubt as to the system of organs to which that particular one, in this special instance, may be considered to belong. Tubercular meningitis has been mistaken for typhoid fever, and *vice versâ;* abscess of the brain has been confounded with malarial poisoning; and, again, tumours in the brain have been regarded as diseases of the stomach; while all the symptoms of cancer of the stomach have been explained away by the fiction of "spinal irritation." Each organ of the body has something in common with every other organ; and although two viscera may be, locally, as remote as the limits of the human body will allow, there are between them bonds of union so intimate that they are, in reality, brought very close together in the minute conditions of pathologic change. Blood, blood-vessels, lymphatics, connective tissue, and nerves are common to all organs which take part in the more active processes of life, and in each of them there is in progress that common nutrition-change which is the necessary condition of all functional activity. Although, therefore, the function of the stomach may be that of exerting a particular effect on food, it must not be forgotten that the stomach has nerves, vessels, and connective tissue, and that diseased processes, exhibiting themselves mainly in its disordered functions, may be the expression of something wrong in innervation, in blood supply, or in general nutrition. And, again, although the brain is the organ which ministers to the higher functions of the nervous system, and records its diseases in changes of mind, sensation, or motility, it must be remembered that the brain has vessels which undergo nutrition-changes of degeneration or decay, and that many of its so-called special diseases are often but the outcome of a more general mal-nutrition, which may have had its starting-point in heart, arteries, or veins. While, then, we may speak of "softening of the brain" among the diseases of the "nervous system," it would be quite as correct, in many instances, to place it among the effects of disease or degeneration in the "vascular system."

All the more important organs of the body are so complex in their structure, and all have so much in common, that we are bound to admit that the lines we draw between them, in regard to pathologic change, are often determined rather by the consideration of their practical utility than by the fact of their scientific accuracy. Knowing, however, the inherent difficulties of the case—viz., those which arise out of the fact that all the systems of organs have marvellously close relations with each other—and being aware, moreover, of those accidental hindrances which arise out of either our own ignorance or want of tact in the application of such knowledge as we have attained, we still hold it to be desirable that, in this System of Medicine, we should maintain the distinction between "general" and "local" diseases, and that we should take as our basis for classification of the latter, the particular systems of organs. We believe this to be so, because the lines which are drawn in making these distinctions include groups of diseases, the individual members of each of which have *inter se* closer clinical relations than have those which belong to different, although contiguous groups; and because in the vast majority of cases it is comparatively easy, and sufficient for all the practical purposes of "classification," to say that a particular disease under consideration is "general" in its character, or is "partial," in the latter instance especially affecting this or that great system of organs, such as the nervous, the respiratory, or the digestive.

The principle of division thus established with regard to all diseases—viz., that of limitation or localisation—is again applicable, as a means to be employed for the primary subdivision of the large group of diseases forming the first section to be considered in this volume; and accordingly "diseases of the nervous system" are distributed under two headings—the "general" and the "special" or "partial."

Under the former, the "general," are described those affections which exhibit their phenomena in all parts of the nervous system—those in which brain, spinal cord, and nerves seem to be *all*, more or less intensely, and more or less widely, involved; while under the latter, the "partial," are detailed those in which the brunt of the malady is borne by particular parts of the nervous system—the brain, the cord, the nerves, or their appendages.

It will be seen at once, by a reference to page 5, that under the former, the "general," are enumerated, together with those which strictly merit that designation, some diseases which appear to affect certain portions of the nervous system either more profoundly or more essentially than they influence others; and further, that some morbid conditions are described which, although limited in the distribution of their symptoms, have as yet no such definite pathological anatomy that we can affirm, with anything like satisfaction, what part of the nervous system is in them primarily at fault. The first large group, therefore, is made to include, together with those in which the whole nervous apparatus is equally disturbed, some diseases apparently partial in their distribution, and others which, in the present state of science, are of "undetermined seat." Among the members of this first large group, for example, there are placed epilepsy, hysteria, and such diseases as show themselves in altered functions of the brain, cord, and nerves—occasionally one great division of the nervous system, and sometimes another, presenting the most marked derangement, but all divisions being more or less involved in morbid change of either function or structure, or both:—and in the same large group we find wasting palsy, chorea, writers' cramp, and allied affections, which, although they exhibit the maximum of their obvious symptoms in particular parts of the nervous system, are yet of such uncertain pathology, that it would be injudicious at present to describe them as diseases of either brain, cord, or nerves, exclusively.

So far as the pathology of these will allow, they will, when examined in detail, be referred to their proper places; but it is thought, for the simple purpose of arrangement, desirable to keep within the limits of ascertained facts and principles, by retaining them in the position already described. The other alternative, that of placing them under particular headings, while it might confer a greater amount of apparent scientific precision, would, I believe, be attended by less real scientific accuracy; inasmuch as it would give an undue prominence to many hypotheses, very valuable in themselves, as forming the framework of both thought and investigation, but which, being as yet no more than hypotheses, are not entitled to hold high rank among the conditions upon which classification should be based. The diseases known, for example, as meningitis, myelitis, and neuritis, respectively, have clinical histories and well-known pathological conditions related to one another in a manner much more definitely ascertained than have such affections as ataxy, paralysis agitans, and wasting palsy. We have referred the symptoms of the first series to their anatomical changes, whereas it cannot be said that we have done so with regard to the second group. Much more is known about the latter than was known a few years ago; but observations, during life and after death, have to be multiplied and verified before we can ascribe those diseases, with scientific precision, to particular localities and special kinds of structural lesion.

Again, the kind of distinction between hæmorrhage into the corpus striatum and hæmorrhage into the spinal cord, is, in its clinical relations, widely different from that which can be established between either of those two affections and ataxy, or chorea. In the one we are dealing with what is definitely known; in the other, with what is as yet indefinite, and only approaching scientific arrangement. Upon these grounds, therefore, the primary division is made into diseases of "general" distribution, or of undetermined locality, and "partial" diseases, or those having a recognised pathological anatomy.

In the second group of diseases of the nervous system—viz. those described as "partial"—the principle of arrangement is sufficiently obvious to need but little elucidation. In the first place, a subdivision is made upon simply anatomical grounds—viz. into affections of the cranium, the spinal column, and the nerves; and each of these is again subdivided upon an anatomical basis—diseases of the cranium being distributed under the categories of "meninges" and "nervous tissues," such as brain, cerebellum, and the like; while diseases of the spinal column are distributed in a similar manner. The next principle of division is that determined by the nature of the anatomical changes which these tissues, respectively, undergo. Here an attempt has been made to place in close proximity those affections which have the most highly-marked clinical similitude—an attempt, however, which is only carried to such a degree as shall not interfere with the more general arrangement.

It is not necessary to enter further into the detail of this classification, as the principles upon which it is based are sufficiently obvious for all the practical purposes of this System of Medicine.

§ I.—Diseases of the Nervous System.

These diseases are divided into two large groups, viz. A, *those of general distribution, or of uncertain seat; and* B, *those which are partial, and which occupy known relations to particular portions of the nervous system.*

A.—General Nervous Diseases, and those of uncertain seat:—

Insanity.
Hypochondriasis.
Hysteria.
Ecstasy.
Catalepsy.
Somnambulism.
Sunstroke.
Alcoholism.
Vertigo.
Chorea.
Paralysis Agitans.
Writers' Cramp.
Convulsions.
Epilepsy.
Locomotor Ataxy.[1]
Muscular Anæsthesia.
Wasting Palsy.
Metallic Tremor.

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[1] Transferred to description of Spinal Cord.

INSANITY.

By Henry Maudsley, M.D., F.R.C.P.

Synonyms.—Insanity; Madness; Mental or Cerebro-Mental Disease; Mental Derangement; Mental Alienation; Mental Aberration; Unsoundness of Mind; Lunacy.

Definition.—So many and various in kind and degree are the forms of mental derangement included under Insanity, that it is not possible to give a definition of it that shall be at the same time comprehensive and exact. If the definition is wide enough to comprise all varieties, it will include eccentricities that fall short of disease; if exact enough to be definite, then it must exclude many cases of undoubted mental disease. As various as are the features or the voices of men, so various are the characters of their minds; and as no two persons are exactly alike in mental character and development, so in no two instances of the degeneration of mind do the morbid features correspond exactly. The development of other organs of the body, taking place before birth after a common type, is very much alike in different persons, and the diseases of them have a great resemblance; but the real development of the brain as the organ of conscious life, taking place after birth in relation to surrounding circumstances, and thus gradually issuing in the formation of individual character, is different in different persons, and accordingly cerebro-mental diseases present manifold varieties of features. As regards any particular case of Insanity which we may have to decide about, it is necessary then to fix attention on two points: first, on the change of individual character—the alteration from the former self; secondly, on the want of harmony, or the discord, between the individual and his surroundings. For although the morbid phenomena of the diseased mind witness in some measure to the degree of its previous development, yet the degeneration which disease implies must needs display itself in an alteration in the kind of manifestation of feeling, thinking, and acting—in other words, in a changed self; while again the import, as morbid, of the phenomena displayed can only be rightly weighed in relation to the individual sphere of life. It is, for example, quite possible, though apt to be forgotten in practice, that sentiments and acts which are habitual in the lowest strata of life may be sure signs of mental disease when uttered and done by one in a high social sphere.

Bearing in mind the difficulties inherent in the nature of the subject, which have led to every sort of definition by every writer who has not forborne the task in despair, I may declare Insanity to consist essentially in a *morbid derangement, generally chronic, of the supreme cerebral centres—the grey matter of the cerebral convolutions, or the intellectorium commune—giving rise to perverted feeling, defective or erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations.*[1] We may safely go so far as to affirm the mind centres to be in the vesicular neurine of the convolutions, and Insanity to consist essentially in disorder, primary or secondary, of their functions—in disordered feeling, disordered intelligence, and disordered will.

This definition has the merit of fixing attention, first, on the reception of impressions from the external world—the mode of *feeling*, or the *affective* life; secondly, on the mental fashioning or elaboration of impressions—the modes of *ideation* or *intellection*, the *intellectual* life; and, thirdly, on the reaction of the individual on the external world—the mode of *action* or *conduct:* it answers also to the best psychological division of mind into *feeling*, *cognition*, and *will*. How desirable it is not entirely to overlook the social relations, will be plain when we reflect that it is in the irregularities of the individual, as an element in the social system, that the morbid character of Insanity fundamentally consists. Certainly the definition is far from being perfect, as in the nature of things must be the case so long as it is impossible to draw the line where sanity ends and Insanity begins, or even to say positively whether a particular person is insane or not; but against its manifest defects may be put its positive merits—namely, that it fixes the grey matter of the convolutions, the undoubted nerve-centres of intelligence, as the principal seat of morbid action in Insanity; that it distinctly declares that Insanity may be exhibited either in moral perversion only, or in the actions of the patient, or in delusion; and, lastly, that it sets forth how Insanity destroys the relations and responsibilities of the individual in the social system, making him very much like what a morbid element is in the organic system—something which cannot take its due place in the general harmony, and which must either be eliminated from it or sequestrated and rendered harmless in it. A man may certainly have disordered feeling, may think and judge erroneously, and act extravagantly, without being insane; but if he does so as a regular thing, and without any adequate cause in external circumstances—if he does so in fact by reason of a steadily acting internal cause, a derangement of his supreme cerebral centres—then he is insane. The standard by which to measure the perversion is, first, that of the *kind*—that which is fixed by the general consent of mankind; and, secondly, that of the

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[1] *Ideation*, now so commonly used, was first suggested and employed by Mr. James Mill in his "Analysis of the Human Mind." Dr. Darwin, in his "Zoonomia," aptly designates the common centres of intelligence as the *Intellectorium commune.*

individual—that which is justified by the degree of his previous mental development.

Many and varied as are the forms which madness takes, there are still beneath superficial differences certain characters of essential agreement; and accordingly genuine groups or types may be described, notwithstanding the fact that cases marking every grade of transition between one group and another are met with in practice. Of Insanity may still be said what Burton long ago said of it: "I could give instances of some that have had all three kinds *semel et simul,* and some successively. . . . . What physicians say of distinct diseases in their books, it much matters not, since that in their patients' bodies they are commonly mixed."

CLASSIFICATION.—The classification commonly adopted in this country, and yet indispensable for practical purposes, is a modification of that proposed by Esquirol, and is as follows:—

I. Mania . . . Acute, or Raving Madness.
  Chronic.
  Recurrent.
II. Monomania.
III. Melancholia.
IV. Moral Insanity.
V. Dementia . Primary.
  Secondary.
VI. Idiocy, including Imbecility.
VII. General Paralysis or Paresis.

In Germany, the classification which finds most favour stands thus:[1]—

I. Die Depressionzustände.	I. Conditions of depression.
1. Die Hypochondrie.	1. Hypochondria.
2. Die Melancholie.	2. Melancholia.
II. Die Exaltationzustände.	II. Conditions of exaltation.
1. Die Tobsucht.	1. Acute Mania.
2. Der Wahnsinn.	2. Monomania.
III. Die psychischen Schwächezustände.	III. Conditions of mental weakness.
1. Die Verrücktheit.	1. Craziness or Incoherence.
2. Der Blödsinn.	2. Dementia or Fatuity.
3. Idiotismus und Cretinismus.	3. Idiocy and Cretinism.
IV. Der paralytische Blödsinn, Die allgemeine Paralysie der Irren.	IV. Paralytic dementia, General Paralysis of the Insane.

It is easy to perceive the defects of such purely psychological classifications. They are vague and artificial, embracing in the same class forms of disease distinct enough to demand a separate description; moreover, there are forms of mental disease which, presenting the characters of two or more of the different classes, might be placed in one or the other, or cannot be placed satisfactorily in either. Dr. Skae has proposed to classify all the varieties of Insanity in natural orders or families, grouping them in accordance with the *natural history* of each.[1] Why, he asks, should we attempt to group and classify them by the *mental* symptoms, and not, as we do in other diseases, by the *bodily disease* of which the mental perversions are but the signs? In pursuance of this aim he has sketched the outlines of twenty-nine natural orders or families, having, as he believes, each its natural history, its special cause and morbid condition, a certain class of symptoms more or less peculiar to each, its average duration, and probable termination. They stand thus:—

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[1] Die Pathologie und Therapie der psychischen Krankheiten. Von Dr. W. Griesinger. Zweite Auflage. 1861.—Die Pathologie und Therapie der psychischen Krankheiten. Von Dr. M. Leidesdorf. Zweite Auflage. 1865.

Idiocy, } Moral and Intel-
Imbecility, } lectual.
Insanity, with Epilepsy.
Insanity of Masturbation.
Insanity of Pubescence.
Satyriasis.
Nymphomania.
Hysterical Mania.
Amenorrhœal Mania.
Post-Connubial Mania.
Puerperal Mania.
Mania of Pregnancy.
Mania of Lactation.
Climacteric Mania.
Ovario-Mania (Utero-Mania).
Senile Mania.
Phthisical Mania.
Metastatic Mania.
Traumatic Mania.
Syphilitic Mania.
Delirium Tremens.
Dipsomania.
Mania of Alcoholism.
Post-Febrile Mania.
Mania of Oxaluria and Phosphaluria.
General Paralysis, with Insanity.
Epidemic Mania.
Idiopathic Mania, { Sthenic.
{ Asthenic.

M. Morel, of Rouen, has propounded a classification of mental diseases according to their apparent causes—an *etiological* classification. He makes six principal groups, each of which has two or three classes under it: the *first* group being that of hereditary Insanity; the *second* consisting of Insanity produced by *toxic* influences; the *third*, of Insanity produced by the transformation of other nervous diseases, such as hysteria, epilepsy, hypochondria; the *fourth*, of idiopathic Insanity; the *fifth*, of sympathetic Insanity; and the *sixth* including all cases of dementia.

Adopting for the purposes of description the classification in common use, artificial as it is, it will be most convenient to describe the special features of the different varieties of mental disease in the course of the account of its causation, symptomatology, and treatment.

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[1] On the Classification of the Various Forms of Insanity on a Rational and Practical Basis, by David Skae, M.D; Journal of Mental Science, October 1863. For a fuller account and a criticism of this classification, I may refer to my work on "The Physiology and Pathology of Mind," Second Edition.

Causes.—These are usually divided into *physical* and *moral*, though without any exactness in such discrimination being really practicable. Two persons are exposed to like severe mental trials: one of them becomes insane, the other does not. Has the madness, then, been produced by a moral cause? In the former case, there was probably some innate vice of nervous element—some predisposition of it to disease, or some accidental nervous depression, by reason of physical disease, or other cause, whereby Insanity has been produced by a moral cause that has had no such ill effect in the latter case. The entire causes have not, then, been in reality the same. What should ever be borne in mind is, that all the conditions which conspire to the production of an effect are alike causes, alike agents, and that there is, in most cases of Insanity, a concurrence of conditions, not one single effective cause. Mental alienation often appears as the natural issue of all precedent conditions of life, mental and bodily—the outcome of the individual character as affected by certain circumstances; in such case, the germs of disease may have been latent in the foundations of the character, and the final outbreak is but the explosion of a long train of antecedent preparations. In vain, then, is it to try to fix always upon a single cause, moral or physical; a common mistake on the part of those who think to do so being to fasten upon that which is in reality an early symptom as the supposed cause. On this rock have hitherto foundered all etiological classifications of Insanity. It will be most convenient to set forth certain general considerations respecting sex, age, and the like, and then to proceed to treat of the proximate or exciting causes of Insanity. It is obviously unscientific to enumerate sex and age as causes of Insanity: no one goes mad because he or she happens to be a man or woman; but because to each sex, and at certain ages, there occur physiological changes that are apt to run into pathological effects in those who are predisposed to nervous derangement.

(*a*) *General Considerations.*—There are general causes, such as the climate of a country, the form of its government and its religion, the state of its civilization, the occupation and habits of its inhabitants, which work together in the course of generations to the formation of a national type of character, wherein there may be greater or less proneness to Insanity. Reliable data respecting the frequency of Insanity in different countries are, unfortunately, still wanting, and even the question whether it has increased with modern civilization has not been positively settled. Travellers certainly agree that it is a rare disease among barbarous people; whilst in the different civilized nations of the world there is, so far as can be ascertained, an average of one insane person in 500 inhabitants. The undoubted steady increase, again, of the insane under care and observation, would seem to be greater than can be fairly accounted for by the greater attention now given to their welfare: while theoretical considerations indicate that the feverish activity of life, the numerous passions and the great strain of mental work incident to the multiplied industries and eager competition of an active civilization, cannot fail to augment the liability to mental disease. Though not yet exactly provable by statistics, there is still some reason to believe that, with the progress of mental development through the ages, there is a correlative degeneration going on, and that Insanity is a penalty which our present civilization necessarily pays.[1]

Sex.—Though Esquirol and Haslam thought Insanity to be a little more frequent among women than among men, it is now generally agreed that the converse is true. Dr. Thurnam affirms men to be more liable to mental disorders than women; and Dr. Jarvis came to the same conclusion from the examination of the statistics of different countries. Recently, however, it has been stated that the female sex is more liable to suffer from hereditary Insanity.[2] Pregnancy, the puerperal state, the catamenial functions, and the climacteric change are conditions in women that will favour the disturbance of the mental balance, especially where there is any predisposition thereto; but against these must be weighed the larger exposure of men to mental wear and tear in the competition of life, and their more frequent addiction to intemperance and other excesses. Women, too, very seldom suffer from general paralysis. On whichever side, male or female, the uncertain difference lies, it is probably inconsiderable.

Period of Life.—Insanity is rare before puberty, though every form of it, except general paralysis, may occur even so early in life; it is far more frequent between the ages of 16 and 25; but it is most frequent of all during the period of full mental and bodily development —from 25 to 45—when there is the widest exposure to its causes. The internal revolution which takes place in women at the climacteric period leads to many outbreaks of Insanity in them between 40 and 50. In men there appears to be a climacteric period between 50 and 60, when Insanity sometimes supervenes: an old man may be found to be keeping a mistress in secret, or to be making foolish proposals of marriage, when, forerunning complete dementia, sensual impulses, clothed in the morbid habit of delusion, mock the extinction of sexual function. In childhood and early life idiocy and imbecility, moral and intellectual, are most commonly met with; after puberty mania and, later on in life, melancholia; in old age senile insanity occurs; and general paralysis seldom before 30, the years between 30 and 50 being the favourite years of its attack.

Condition of Life.—The statistics hitherto collected in regard to this point are of little or no value. Whether a particular profession or trade favours the production of Insanity is generally a question of the habits incidental to its pursuit—whether those who follow it live soberly and temperately, or whether they are addicted to intemperance and riotous living. On the whole, however, those who work with the head are more liable to mental disease than those who work with the hand, and they are less liable to recover when once attacked. It is an unproved and indeed ill-founded assertion that governesses are the victims of Insanity in greater proportion than other persons. The statement has originated in the fact that a great number of governesses are received into Bethlehem Hospital, as many as 110 having been admitted in ten years. The reason of this is that Bethlehem is intended especially for persons of the class of governesses—those who are not paupers, but yet cannot pay for care and treatment.

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[1] In 1859 the total number of lunatics in England and Wales was 36,762; in 1869 it had increased to 53,177. The proportion of lunatics to the population had risen from 1 in 536 to 1 in 411 of the population. In France the ratio of lunatics to the population was, in 1851, 1 to 796; and in 1861, 1 to 444. In both countries, however, it is certain that the main portion, if not all, of the increase has been due to the operation of the lunacy laws, by which more accurate registrations of the insane have been gradually brought about.

[2] Statistics of Insanity of the Crichton Royal Institution, by H. G. Stewart, M.D.; Journ. Ment. Science, 1865. Also, Hereditary Insanity, by H. G. Stewart, M.D.; Journ. Ment. Science, 1864.

Other things being equal, it is certain that Insanity is proportionately more frequent amongst the unmarried than the married.

Individual predisposition.—The heritage which a man has from his parents may alone, or together with the circumstances of early education, give rise to an individual predisposition to mental derangement. Unquestionably some persons have what may be called the insane temperament—a certain *neurosis* or *diathesis*, easily prone to degenerate into actual disease; they feel impressions in a way which other people do not feel them, are disposed to sudden impulses of strange feelings and desires, to whimsical caprices of thought and eccentricities of action, and they not unfrequently carry in their countenance and bearing the marks of their evil heritage. They have what Willis long ago called the *diathesis spasmodica*, an irritable weakness of nervous constitution, in which, if there be not positive disease, there is the well-prepared ground of disease. Authors are not agreed as to the proportion of cases of Insanity in which positive hereditary taint is detectable: some, like Moreau,[1] putting it as high as nine-tenths; others, as low as one-tenth. The most careful researches fix the proportion as not lower than one-fourth, if not so high as one-half; and there can be no doubt that the tendency is to increase the proportion as investigation becomes more searching and exact. When a person cannot endure the ordinary trials of life, or a natural physiological function, such as the development of puberty, it is plain that there must be some native infirmity or instability of nerve element. It must be borne in mind that hereditary predisposition may be of every degree of intensity, so as, on the one hand, to conspire only with certain more or less powerful exciting causes, or, on the other hand, to suffice of itself to give rise to Insanity even amidst the most favourable external circumstances. Again, not Insanity only in the parents, but any form of nervous disease in them—epilepsy, alcoholism, hysteria, and even neuralgia—may predispose to Insanity in the offspring, as, conversely, Insanity in the parent may predispose to other kinds of nervous disease in the offspring. Procreation during the temporary insanity of drunkenness, and too much interbreeding in families, are both recognised causes of a predisposition to mental degeneration. Some, like Lugol and Schroeder van der Kolk, have maintained that scrofula of parents may generate a predisposition to Insanity in the children; and whether this be so or not, it can admit of no question that the undoubted transformation which diseases undergo through generations is a subject deserving of further and more exact study.[1] Baillarger has proved, what Esquirol observed, that Insanity descends more often from the mother than the father, and from the mother to the daughters more often than to the sons. Children born before the outbreak of an attack are less likely to suffer than those born after an outbreak.

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[1] Psychologie Morbide dans ses Rapports avec la Philosophie de l'Histoire. Par Dr. J. Moreau.

An injudicious *education* may aggravate an inherent mischief; the parent not only transmitting a taint or vice of nature to the child, but fostering its increase by the influence of a bad example, and by a foolish training at that period when the young mind is very susceptible, and the direction given to its development decisive for life. Where there is no innate taint, mischief may still be wrought by enforcing an unnatural precocity, wherein is often planted the germ of future disease. Parental harshness and neglect, repressing the child's feelings, stifling its need of love, and driving it to a morbid self-brooding, or to take refuge in a world of vague fancies, is sometimes not less injurious than a foolish indulgence, through which it never learns the necessary lessons of renunciation and self-control. There can be no doubt that by the influence of good education and sound training a predisposition to Insanity might often be much neutralised and rendered almost harmless; but the mischief is that those who procreate children so afflicted are commonly least fit to train them well.

(*b*) *Exciting Causes.*—The so-called *moral* causes are generally, though not universally, held to be more frequent than the *physical* causes: Pinel thought them to be twice, Esquirol four times, as frequent; while Guislain attributed 66, Parchappe 67, out of 100 cases of Insanity to moral causes.[2] It is not the way of great intellectual exercise, when unaccompanied by emotion, to lead to mental derangement; mental exercise is favourable to length of days and health of mind; it is when the feelings are deeply engaged, when the mind is the theatre of great passions, that it is most moved and its stability most endangered. The depressing passions are most effective in this regard: grief, religious anxiety, disappointed affection or ambition, jealousy, the wounds of an exaggerated self-love, and the painful feeling of being unequal to responsibilities, or other such conditions of mental agitation and suffering, are most apt to reach a violence of action by which the balance is lost. It is especially when the individual has by a long concentration of thought, affection, and desire on a certain aim or object grown into definite relations with regard to it, and made it, as it were, a part of the inner life, that a sudden and entire change, shattering long-cherished hopes, is most likely to produce Insanity; for nothing is so fraught with danger to the stability of the strongest mind as a sudden great change in external circumstances without the inner life having been gradually adapted thereto. Hence, also, it is that a great exaltation of fortune, as well as a great affliction, rarely fails to affect for a time the strongest head and sometimes quite overturns a weak one; though the strong mind succeeds after a time in establishing an equilibrium between itself and its new surroundings, which the feeble mind cannot do. Men do not, however, often become insane from joy; and when one of the expansive passions, as ambition, religious exaltation, overweening vanity in any of its Protean forms, leads to mental derangement, it does not, like a painful passion, act either directly as the sudden cause of an outbreak, or indirectly by producing organic disorder and subsequent Insanity, but it exhibits its effects slowly, as a gradual development or exaggeration of a particular vice of character.

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[1] Die Pathologie und Therapie der Geisteskrankheiten auf anatomisch-physiologischer Grundlage. Von J. L. C. Schroeder van der Kolk. 1863.

[2] Pinel, On Insanity, translated by Dr. Davis; Esquirol, Traité des Maladies Mentales; Guislain, Traité sur l'Aliénation Mentale; Parchappe, Traité de la Folie.

Among the causes of mental disturbance which it would be difficult to pronounce other than moral, but which are really due to physical conditions, are those incident to the great mental revolution produced by the development of the sexual system at puberty; when there occurs, as Goethe aptly expresses it, "an awakening of sensual impulses which clothe themselves in mental forms, of mental necessities which clothe themselves in sensual images." The great moral commotion produced at this period is the cause of an unstable equilibrium of mind, which is just as dangerous as if it were produced by some external cause; and which, if hereditary predisposition exist, may, without further auxiliary cause, issue in Insanity.

Of the *physical* causes of Insanity, intemperance occupies the first place; acting not only as a direct cause, but indirectly through the emotional agitations incident to an irregular life of dissipation and excess. Opium, Indian hemp, and other narcotics notably give rise to temporary disorder of mind, and, if abused by long indulgence, they may lead to permanent degeneration. Self-abuse in men is the cause of a particularly disagreeable form of Insanity, characterised by intense self-feeling and conceit, indolence and vacillation of character, and profound moral disturbance in the earlier stage, and later, by failure of intelligence, nocturnal hallucinations, and suicidal or homicidal propensities. Epilepsy is sometimes followed by a most violent and dangerous mania, and, when of long standing, produces loss of memory, and general failure of intelligence. Sometimes an outbreak of mania precedes or takes the place of an epileptic attack; and it may happen that a painful form of moral derangement, with periodical exacerbations —a masked epilepsy—precedes for months the appearance of the genuine epileptic convulsions.

In some instances hysteria produces or passes into Insanity. An attack of acute maniacal excitement, with great restlessness, rapid and disconnected, but not entirely incoherent, conversation, sometimes tending to the erotic or obscene, evidently without abolition of consciousness; laughing, singing or rhyming, and perverseness of conduct, which is still more or less coherent and seemingly wilful,—may occur in connexion with, or instead of, the usual hysterical convulsions. Or the ordinary hysterical symptoms may pass by degrees into chronic Insanity. Loss of power of will is a characteristic symptom of hysteria in all its Protean forms, and with the perverted sensations and disordered movements there is always some degree of moral perversion. This increases until it swallows up the other symptoms: the patient loses more and more self-control, becoming capriciously fanciful about her health, imagining or feigning strange diseases, and keeping up the delusion or the imposture with a pertinacity that might seem incredible, and getting more and more indifferent to and impatient of the advice and interference of others. Outbursts of temper become almost outbreaks of mania, particularly at the menstrual periods. An erotic tinge is sometimes observable in the behaviour.

More or less dulness of intelligence and apathy of movement, giving the seeming of a degree of imbecility, is common enough in chorea, and in some cases there is a violent delirium or mania; but besides these cases there are, I believe, in children others in which, without disorder of movements, there is a true choreic mania: it is an active delirium of ideas which is the counterpart of the usual delirium of movements, and its automatic character and marked incoherence are very striking; hallucinations of the special senses, and loss or a perversion of general sensibility usually accompanying delirium.

Chronic diseases, constitutional and local, favour the production of Insanity in many instances. Anæmia plays the same weighty part as in the causation of other nervous diseases. It is not without influence in many cases of hysterical insanity, as well as in the asthenic form which occurs during lactation; and when suddenly produced by great loss of blood, it may be the cause of an attack of puerperal mania. The syphilitic virus is now known to affect nervous element injuriously, and of late an extreme form of dementia has been ascribed to a syphilitic exudation, circumscribed or diffused, on the surface or within the substance of the brain. Tuberculosis is frequently associated with mental disease, one-fourth of the deaths in asylums being due to phthisis; and a form of suspicious melancholia, having something of the character of dementia about it, has been described as phthisical nsanity.[1] The disappearance of a skin disease, or the suppression of an accustomed discharge, has of old been known to be at times followed by an attack of mania or melancholia; and there are on record numerous cases of mania which have been caused by retrocedent gout. Of local diseases favouring the production of Insanity, the influence of those of the heart seems to have been overrated; out of 602 post-mortem examinations made in the Vienna asylum, the heart was found to be affected in one-eighth, and in some of these only very slightly. Abdominal diseases are sometimes genuine causes of melancholia; and diseases of the sexual organs in women have always had a high place assigned to them in the scale of causes. It is certain that an attack of mania has followed the suppression of the menses, and that the return of menstruation is often followed by the recovery from Insanity; but it is certain also that outbreaks of maniacal fury, or of suicidal or homicidal violence, have coincided with the period of menstruation. Schroeder van der Kolk had a patient profoundly melancholic, who suffered also from prolapsus uteri, and in whom the melancholia disappeared directly the uterus was restored to its place. Flemming relates two similar cases in which the melancholia was cured by the use of a pessary, in one of them regularly returning whenever the pessary was removed; and I have seen, in one case, severe melancholia of two years' duration disappear after the cure of a prolapsus uteri. Instances are on record in which a woman has regularly become insane during each pregnancy; and, on the other hand, Guislain and Griesinger mention a case, respectively, in which Insanity disappeared during pregnancy, the patient at that time only being rational.

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[1] Tuberculosis and Insanity, by T. S. Clouston, M.D.; Journ. Ment. Science, April 1863.

Under the name of Puerperal Insanity are frequently confounded three distinct varieties—the Insanity of Pregnancy, Puerperal Insanity proper, and the Insanity of Lactation. The first and last usually have the form of marked melancholia with suicidal tendency; the second appears as an acute and incoherent mania.

The Insanity which sometimes breaks out at the change of life in women is commonly a profound melancholia, with vague delusions of an extreme character.

After acute febrile diseases, as typhus and typhoid fever, the acute exanthemata, acute rheumatism, and pneumonia, Insanity sometimes follows. In such cases it either takes the form of acute dementia, or of the mild delirium of nervous exhaustion, from which recovery takes place in a few days; or it steadily passes into a chronic and persistent form, especially if there be hereditary taint; or it is acute, recovery taking place for a time, but, as happens after injuries to the head, being followed by subsequent marked change of temper, and finally Chronic Insanity.[1]

Injuries of the head, when not followed by any immediate ill consequences, may still, after a time, lead to incurable Insanity, through the degenerative changes which they induce in the cortical layers. Insolation notably acts perniciously on the cerebral centres, either by causing acute hyperæmia, and œdema, or, as is more probable, by overstimulation and consequent exhaustion of nervous element. Abscesses and tumours of the brain, cysticerci,[2] effusions of blood, do not directly produce mental disorder, which is indeed often absent; and when they do give rise to such disorder, they seem to act indirectly by a reflex or sympathetic action. Professor Gerhardt relates one case in which mental derangement was the first symptom of an embolism, the paralytic phenomena following later; and in a case recorded by L. Meyer, chronic tubercular meningitis gave rise to mental disorder.[1] Instances are on record in which Insanity, like tetanus, has been caused by peripheric injury of nerve; and of great interest are those cases, long since observed by Dr. Darwin, in which it occurs as the transference of disorder from the spinal centre.

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[1] De la Folie Consecutive aux Maladies Aigues. Par le Dr. E. Muguier. Paris, 1865.—Griesinger, op. cit.—On the Delirium of Acute Insanity during the Decline of Acute Diseases, by Hermann Weber, M.D.; Med.-Chir. Trans. 1848.

[2] On Cysticerci, Archiv der Heilkunde, 1862, Prof. Griesinger. A case of insanity in which several cysticerci were found in the brain is related by Joire in the Gazette des

Let it be distinctly understood, however, that of the above enumerated causes of Insanity, it scarcely ever happens that one acts singly; many of them would have no such ill effect, except through the co-operation of hereditary predisposition, and the latent hereditary taint might remain happily dormant, but for the concurrence of unfavourable conditions, physical or moral. Whenever such inborn taint does exist, it is certain that any great revolution in the system, whether arising out of external circumstances, or from internal causes, such as puberty, pregnancy, and the climacteric period, will be fraught with danger to the healthy balance of the mind.

FORMS OF INSANITY AND THEIR SYMPTOMATOLOGY.—A glance at the symptoms of the various forms of mental disease reveals at once the existence of two well-marked groups: one of these including all those cases in which the mode of *feeling* or the *affective* life is chiefly or solely perverted—in which the whole habit or manner of feeling, the mode in which the individual is affected by events, is entirely changed; the other, those cases in which *ideational* or *intellectual* derangement predominates. More closely scanning the symptoms, it is seen that the affective disorder is the fundamental fact; that in the great majority of cases it precedes intellectual disorder; that it co-exists with the latter during its course; and that it frequently persists for a time after this has disappeared. Esquirol rightly, then, declared "moral alienation to be the proper characteristic of mental derangement." "There are madmen," he says, "in whom it is difficult to find any trace of hallucination, but there are none in whom the passions and moral affections are not perverted and destroyed. I have in this particular met with no exceptions." This experience is in entire accord with that of every observer of Insanity, and with the principles of a sound psychology. It is the feelings that reveal the genuine nature of an individual; it is from their depths that the impulses of action come, while the intellect guides and controls; and accordingly in a perversion of the affective life is revealed a fundamental disorder of the innermost nature, a dis-

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Hôpitaux, 1860; another by Dr. Snell in the Zeitschrift für Psychiatrie, 1861; another by Baillarger in the Arch. Clin. des Maladies Mentales, 1860; and another by Dr. Saunders in the Report of the Devon County Asylum for 1864.

[1] Prof. Gerhardt, Wiener Med. Presse, No. 7, 1865; L. Meyer, *Zeitschrift für Psychiatrie*, 1858, p. 716.

order which will be exhibited in acts, rather than, as ideational disorder is, in words. To insist upon the existence of a delusion as a criterion of Insanity, is to ignore some of the gravest and most dangerous forms of mental disease.

Melancholia.—Here the fundamental fact is a deep, painful feeling of profound depression and misery, a great mental suffering. The patient's feeling of external objects and events is perverted, so that he complains of being strangely and unnaturally changed: impressions which should rightly be agreeable, or only indifferent, are felt as painful; friends and relatives are regarded with sorrow or aversion, and their attentions with suspicion; he feels himself entirely isolated, and can take no interest in his affairs; and he either shuns society and seeks solitude, lying in bed and unwilling to exert himself, or he utters his agony in sounds ranging from the moan of dull ache to the shrill cry of anguish, or in ceaseless gestures of misery, or even in some convulsive act of desperate violence. All this while there may be no delusion; the patient may be conscious of the change in himself, may grieve over his unnatural state, and strive to hide or fitfully resist it: but as he gets worse he becomes more and more self-absorbed, more and more indifferent to, or distrustful of, those around him, and, finally, succumbs entirely to his affliction. Then it is, usually, that the vast and formless feeling of profound misery takes form as a concrete idea —in other words, is condensed into some definite delusion: this now being, as it were, the expression of it. The patient believes that he has committed some great crime, for which he must suffer death on the gallows; that he has blasted the happiness of his family; that he is possessed by the devil, or is the victim of a persistent and cruel persecution, by magic or by magnetism; that he has committed the unpardonable sin, and is for ever damned. The delusion is not the cause of the feeling of misery, but is engendered of it, and takes different forms according to the degree of the patient's culture, and the social, political, or religious ideas prevailing at the particular epoch: what the uneducated person attributes to witches or to devils, the man of some cultivation attributes to magnetism or to political conspiracy. In certain cases it is striking how disproportionate the delusion is to the extreme mental anguish—how inadequate it is as the expression of it: one, whose agony is that of the damned, will aver that it is because he has drunk a glass of beer which he should not have done, or because he has muttered a curse when he ought to have uttered a prayer. With him who believes that he is doomed to infinite and eternal misery, it is not the delusion, but the affective disorder, that is the fundamental fact; there can be no adequate or definite idea of the infinite or eternal, and the insane delusion of eternal damnation is but the vague and futile attempt at expressing an unutterable real suffering. It is noteworthy, again, how much the affliction of the melancholic subsides sometimes when a definite delusion is established: the vast feeling of vague misery which possessed the whole mind has undergone systematization in definite morbid action; and when the delusion is not active, but reposes in the background, not otherwise than as ideas constantly lie dormant in the sound mind, the patient may be tolerably cheerful. A suicidal feeling is so common that the possibility of its existence should always be had in mind; in 51 cases of Insanity in which suicide had been meditated or attempted, 28 were cases of melancholia.

As many as are the varieties of mental pain or suffering, so many varieties are there of melancholia; the essential character of all of them being an oppression of the self, the weight of a great suffering, out of which springs the delusion of being overpowered by some external agency, demoniac or human, or of salvation lost through individual sins. The classification of melancholia according to the accidental character of the delusion is, therefore, of little value. Two well-marked groups may be distinguished: the first, including those who have a definite delusion—*Lypemania;* the second, those who have no definite cause of terror, but display a fearful apprehension of everything possible and actual—*Pantophobia.*

Hypochondriacal Melancholia represents one of the mildest but most persistent forms of melancholic depression; the anxiety proceeding from an extravagant feeling of bodily disease and exaggerated notions of danger. The morbid feeling, which is not usually without some physical cause in the organism, may be general, or it may be confined to single anomalous sensations. The patient is anxious and depressed; he complains of anomalous feelings, which he watches and analyses very attentively; his heart flutters fearfully, a film passes over his eyes, and there are strange sensations in his head; he examines his pulse, tongue, and evacuations, and rarely fails to find something abnormal in all of them. He is commonly irresolute, sluggish and indifferent to what is not related to the circle of his morbid ideas; but in some cases paroxysms of anguish and despair rise to such a height as to sweep away all power of self-control, and to issue in suicidal or homicidal violence. The intelligence, though generally sound in regard to all matters that are not overclouded by the morbid feelings, is still profoundly affected through these. Hence, though hypochondriacal melancholics do not often commit suicide or homicide, they may do both; a man in the Somerset Asylum, for example, cut into his belly with a piece of glass, and dragged out his small intestines, in order to let the wind out. The transition is indeed gradual from the less severe forms to those in which the anomalous sensations are not merely exaggerated and misinterpreted, but are referred to some absurdly unreal cause, as to the presence of a serpent in the stomach, or to a galvanization of the nerves, or even to those cases in which the patient supposes his legs to be glass, his body butter, or himself metamorphosed into a wolf. By this declension, hypochondriacal melancholy undoubtedly passes into true melancholia.

Climacteric Insanity usually takes the form of profound melancholia, with vague and vast delusions, as that the world is in flames, that it is turned upside down, that everything is changed, or that some very dreadful but undefined calamity has happened or is about to happen. The countenance has an expression of a vague terror and apprehension. In some cases short and transient paroxysms of excitement break the melancholy gloom; these usually occur at the menstrual periods, and may continue to do so for some time after the function has ceased.

In connexion seemingly with the development of puberty, or at any rate soon afterwards, we sometimes meet with a fanciful and *quasi-hysterical melancholia* in girls, which is not very serious when it is properly treated. There are periods of depression and paroxysms of apparently causeless weeping, alternating with times of undue excitability, more especially at the menstrual periods; a disinclination is evinced to work, to rational amusement, to exertion of any kind; the conduct is capricious and soon becomes perverse and wilful; the natural affections seem to be blunted or abolished, the patient taking pleasure in distressing those whose feelings she would most consider if in health; and although there are no fixed delusions, there are unfounded suspicions or fears and changing morbid fancies. In some of these cases, when the disease has become chronic, delusions of sexual origin occur, and the patient, whose virginity is intact, imagines that she is pregnant or has had a baby.

The *Insanity of Pregnancy* is, as a rule, of a marked melancholic type, with suicidal tendency; a degree of mental weakness or apparent dementia being sometimes conjoined with it. Other cases, however, exhibit much moral perversion, perhaps an uncontrollable craving for stimulants, all which we may perhaps regard as an exaggerated display of the fanciful cravings, the capriciousness and the morbid fears from which women suffer in the earlier months of pregnancy.

The *Insanity of Lactation* is an asthenic melancholia, often with determined suicidal tendency. The time of its occurrence seems to show that the longer the child is suckled, the greater is the liability to it.

Sensibility is commonly much affected in melancholia. There may be a general diminution of the sensibility of the skin, or a local complete loss thereof; and complaints of præcordial anguish and of strange abdominal sensations testify the perversion of organic sensibility. These anomalous sensations appear sometimes to have a relation to the confusion and anguish of mind not unlike that which the epileptic aura has to the epileptic fit. Illusions and hallucinations of the special senses are frequent: the patient seeing those around him as devils, or smelling a corpse in his room, or tasting poison in his food, or hearing voices which revile and accuse him, or which suggest impious thoughts and prompt to violent deeds—it may be to imitate Abraham, and sacrifice his child.[1]

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[1] If a person sees, hears, or otherwise perceives what has no existence external to his senses, he has a *hallucination;* if he sees, hears, or otherwise perceives that which has no such external existence as he perceives, or perceives it with erroneous form or qualities,

The bodily nutrition usually shares in the general depression of tone, although it is sometimes remarkable, considering the great apparent suffering, how little it is affected. When it does suffer, digestion fails, and constipation is troublesome; the skin loses its freshness, becomes sallow, dry, and harsh; the temperature of the body is lowered, and the extremities are cold; the respiration is slow, moaning, and interrupted by deep sighs; the pulse is feeble, sometimes very slow, and even intermittent; and menstruation is generally irregular or suppressed. Sleep is usually deficient, though patients are apt to assert that they do not sleep when they really do, so little do they feel refreshed by it. Refusal of food, which is common, and sometimes very persistent, may be due to other causes besides want of appetite: it may take place through a fear of being poisoned, or in consequence of a delusion that the intestines are sealed up, or in order to commit suicide by starvation, or in fancied obedience to a voice from heaven, or from sheer wilful obstinacy.

The behaviour of the melancholic accords with, or fitly expresses, the character of his ideas and feelings; and three well-marked groups of melancholia may be made according to the different relations on the motor side:—

1. Melancholia with stupor, *M. attonita*, is interesting because of its close resemblance to dementia, with which it has been confounded. The expression of the face is that of a vacant, self-absorbed amazement, or the fixed form of some painful passion; the patient, as if in a trance, or as one only partially awake, scarce seems to see or hear; there is partial or general insensibility of the skin; consciousness of time, place, and persons is lost, and the bodily wants and necessities are alike unheeded; the muscles are generally lax, or some of them are fixed in a cataleptic rigidity. The patient—who, statue-like, must usually be removed from place to place—is possessed with some terrible delusion, as that the whole world is in flames, or that he is standing on the edge of a sea of blood, and when he recovers his senses he is as one awakened out of a frightful dream. One lady under my care, who was in this state for two years, with the exception of an occasional break of lucidity for a few hours, and who ultimately recovered her senses quite suddenly, believed that every one who approached her, and even lifeless objects, were threatening to murder her. As may easily be imagined, it is not always possible to distinguish this condition from dementia; for as to live in one sensation would be equivalent to having no sensation at all, so for a mind to be entirely absorbed in one terrible delusion, to remain in one persistent state of morbid consciousness, is equivalent, for the time being, to there being no mind at all. As, however, recovery may take place rather

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he has an *illusion*: and if, though perceiving external objects as they really exist, he believes in the existence of such objects, or conceives such notions of the properties and relations of things, as are absurd to the common sense of mankind, he has an insane conception or a *delusion*—the ground of the falseness of conception being not error, but a morbid condition.

suddenly, though it may sometimes last only for a few hours or days and then be followed by a complete relapse, it is plain that melancholia with stupor is different from the stupor of real dementia.

2. Melancholia is often accompanied with destructive impulses, to sudden acts of violence against self or against others. Suicidal impulse is very common amongst melancholics, some sincerely and bitterly grieving over the horrible propensity as the sole cause of their unhappiness; but what is very remarkable is the sudden manner in which patients usually calm are at times surprised and overpowered by a desperate impulse and hurried into a convulsive act of violence. A quiet man, having the delusion that his soul was lost, who had been for months under my care, and of whom no one suspected any mischief, suddenly started out of bed one night, without any warning, and flung himself out of a window through which it would have been thought impossible that any man could get. He was possessed with terrible hallucinations, thought that the world was come to an end, and in a fearful state of writhing agony cried, "Let me go! let me go!" Like paroxysms recurred occasionally during the next few weeks, after which the man recovered. The time of waking from sleep is that at which the desperate impulse is most likely to arise, wherefore melancholics should never be left alone when getting up in the morning. In other cases the sudden act of violence may be directed against others; the patient injuring or killing some one by reason of a sudden hallucination, or in consequence of his anguish having reached such a height of unendurable agony as to abolish all self-control, and irresistibly to utter itself in convulsive violence, either against a fancied persecutor or a completely indifferent person (*Raptus melancholicus*). Of such are some homicidal lunatics. Others act in obedience to a delusion: an evil spirit instigates the demonomaniacs to desperate deeds in spite of the will, or its impulses intensify their misery and lead to determined suicidal attempts, in order to escape from the intolerable promptings. A melancholic mother has killed herself to escape the desperate impulse to kill her child. Nor is it inconsistent with insanity in such cases that the violent deed should have been planned with surprising cunning and effected with sustained ingenuity.

So far from the morbid impulse or act constituting the insanity, it is but the outward and visible sign or expression of a profound affective derangement, the tendency of which is to manifest itself, not as ideational insanity does, in words, but in acts, and which for this very reason is much more dangerous than ideational insanity: it is truly an affective insanity, one symptom of which is homicidal or suicidal impulse; the delusion, when there is one, and the homicidal act, are both symptoms of a deeper-lying disease; and the morbid manifestation of one may be as little within control as that of the other, or as the suddenly arising hallucination is. In the one case the patient is the victim of a morbid idea; in the other, of a morbid movement—in both cases, of a convulsion more or less co-ordinated. Where the disease is less acute, it is the feeling of this affective perversion that sometimes drives the melancholic to commit murder in order to be hanged, or impels a mother to murder her children in order to send them from misery on earth to happiness in heaven. It admits of no question whatever, and should therefore be borne clearly in mind, that the calmest melancholic is liable to occasional unaccountable exacerbations of disease, during the paroxysms of which he may perpetrate violence against himself or others; a wonderful relief, and even an apparent sanity, with endeavour to escape penal consequences, sometimes following the accomplishment of the act.

3. There is a melancholia of acute character, with great excitement and restlessness, that may even pass into mania. It is certain that cases marking every step of the transition to mania do occur in practice; and it is not always easy, notwithstanding the painful character of the delusion, to distinguish excited melancholia from mania: there are truly melancholics who are maniacal, as there are maniacal patients who are melancholic. The more activity of movement there is as the expression of the mental suffering, the more acute the utterance of the agony in gesture-language—in the wringing of the hands and the writhing of the body—the nearer does the case approach mania. The manifestations of excitement are, however, generally of a more uniform character than those of mania, and often even monotonous.

The *course* of melancholia is generally chronic; remissions are common, but complete intermissions rare. Still, it is striking sometimes how suddenly a great change may take place: Griesinger mentions a case of deep melancholia in which there occurred a perfect lucid interval for the space of a quarter of an hour; and I have more than once seen a profound melancholic awake in the morning cheerful and seemingly quite well, remain so for the rest of the day, and yet be as bad as ever on the following day. Such sudden recoveries are, like sudden conversions, greatly to be distrusted. Still, I have met with two instances in which sudden recoveries were permanent: in one, the patient, who had been acutely melancholic for six months, recovered suddenly after a flood of tears; in the other, the patient was quite well in the morning, after a sleepless night of much mental anguish and excitement. When recovery really takes place, it is usually gradual, and from within four to twelve months from the commencement of the disease; it is rare, but not impossible, after a year, although it may occur now and then after several years, especially if some great shock has aroused the patient to exertion. Half, or even more than half, of the cases of melancholia get well under proper treatment; and of those which do not recover, about half decline into mental weakness or complete dementia—the rest remaining chronic or ending in death. Death may be caused directly by the exhaustion of excitement or refusal of food, or it may be due to intercurrent diseases, phthisical, cardiac, or abdominal. Gangrene of the lung was found by Guislain most frequently in melancholics who had died after long refusal of food.

Mania.—In this form of mental disease there is an excitement or exaltation of the self-feeling of the individual, the expression of which takes place either in the movements and conduct or in the character of the thoughts. Accordingly, two groups of cases may be broadly distinguished, although they pass insensibly into one another and are not unfrequently mixed; the first including all those cases of acute mania or maniacal fury in which the madness is mainly manifest in the actions of the patient, who sings, dances, declaims, runs about, pulls off his clothes, and in all ways acts most extravagantly; the second group including those more chronic cases in which the derangement is expressed in the ideas, is systematized in definite delusions—in which, therefore, the morbid action has taken deeper hold of the individual. The first group corresponds in the main to *acute mania*, the second to *monomania.*

It was held by Guislain that a stage of melancholic depression, of greater or less duration, almost invariably precedes an attack of mania; and there can be no doubt that this sequence is traceable in many cases. But it is not so in every case, as some have maintained. What has been commonly overlooked is, that there is not only an affective disorder of a depressed or melancholic kind, but that there is also an affective Insanity which is rather of an excited, expansive, or maniacal kind—a deep derangement of the affective life, in which the individual's self-feeling is greatly exaggerated or morbidly exalted, without positive intellectual alienation. It is a maniacal disorder, so to speak, of the feelings, sentiments, and acts, without delirium; and it is expressed, as the corresponding affective melancholia is, not in delusion, but in the conduct of the patient. As it is from the affective life that the impulses of action come, while the function of the intellect is to guide and control, it is in strict accordance with reason that, when there is affective derangement or perversion of the mental tone, the morbid impulses that arise should be beyond control or guidance of the will, just as the convulsion of a limb is beyond control when there is derangement of the tone of the spinal centres. This inceptive maniacal state, which may unquestionably be primary, though usually following that of melancholic depression, is displayed in a great change of moral character; the parsimonious becomes extravagant, the modest man presumptuous and exacting, and the affectionate parent indifferent to his family; there is an extreme liveliness of manner, or a restless and busy activity, as of one half intoxicated; an overweening self-esteem is a marked feature, and an extravagant expenditure of money or an excessive sexual indulgence is common. Or the *exaltation* may be less and the *perversion* of the affective life more marked; in other words, the moral *alienation* more extreme, as witnessed in the profound moral derangement which sometimes precedes a series of epileptic fits, or takes the place of an epileptic fit, and in most of those cases included by Pinel under *mania sine delirio* and by Prichard under *moral insanity*. In such cases, as with the cases of so-called irresistible homicidal or suicidal impulse, it has been too much the practice to fix attention exclusively upon the extravagant actions of the patient, to the neglect of the profound affective derangement out of which his acts spring; so that they have been set apart as special and their real relations overlooked. They are truly of the same nature as that maniacal perversion of the whole manner of feeling sometimes forerunning an outbreak of mania; and their morbid expressions in single acts of vicious or violent conduct are of the same kind as those general symptoms of acute mania which are exhibited in the movements or actions of the patient.

Acute Mania; Maniacal Fury or Frenzy; or Raving Madness.—It seldom breaks out without a preceding stage of affective derangement, the period of incubation being usually of a melancholic character, as though there were a painful forefeeling of the coming storm. After a shorter or longer period of such premonitory depression there follows a marked change in the inclinations, habits, and affections: the patient, "much, much different from the man he was," gets restless, and is prone to wander or travel about, is sleepless at night, or is tormented with very vivid dreams; he next becomes lively and excitable, as though half intoxicated, and the tone of his voice is sometimes strangely altered; his actions are restless, extravagant, and turbulent; and all the while he thinks himself wonderfully well, and scorns the suggestion of medical aid. As matters become worse there is an irresistible propensity to utter the internal commotion in outward gestures, acts, or words: the patient sings, dances, declaims, shouts, and laughs; or he is industriously occupied in restless and aimless work, as in polishing the floor with his saliva, in tearing his clothes to shreds, or in changing the place of every piece of furniture about him; or he explodes in furious outbreaks of rage and raving of word and action. The organic appetites or instincts come markedly into the foreground, the veil of reason being withdrawn: the appetite is ravenous and indiscriminate, garbage, or even excrement, being devoured with apparent avidity; and the patient, forgetful of decency, and abandoned to the promptings of the sexual impulse, sometimes masturbates as the monkey does, without shame or restraint. Withal there is often a certain consciousness of his state, so that he may restrain himself and seem reasonable for a time, and when seemingly at his worst he will sometimes yield to the show of energy and determination. The mood of mind may be brisk and humorous, or bitter, angry, and scornful in the face of opposition. There is no fixed delusion, nor any fixed group of delusions; but the ideas are rapid, confused, and transitory, and appear as fleeting delusions, or immediately utter themselves in automatic impulses to words and actions: the idea of an act, the moment it arises in the mind, becomes the act. Because of the rapidity of the flow of ideas in the early stages, the witty observations, acute comparisons, and fluent rhymes then sometimes made, it has been said that there is an increase of mental power. But it is only the semblance of an increase: though there is a lively revival of the past with great vivacity of expression, there is no due assimilation of the present, but an incapacity to perceive rightly the relations of things around, together with false judgment with regard to them, so that the unhappy sufferer is extravagantly joyous in a madhouse; there is an entire absence of that co-ordination of the feelings and ideas which marks the highest mental power and is the condition of true will. The lively flow of scarce coherent ideas marks the excitability of an irritable weakness, and is the forerunner of a restless succession of isolated ideas and fragmentary associations in the more advanced stages, not otherwise than as convulsion is the forerunner of paralysis.

It is striking how complete in some cases may be, during the attack, the memory of the past, and, after the attack, of all that has happened during it; whilst, in other instances, the patient will forget altogether the events of his madness, like as a dream is forgotten, though he may remember them again during a subsequent attack. It may happen also that, immediately before a second attack, thoughts and feelings displayed on the occasion of a first attack, but latent since, will recur, so that even attendants recognise the evil presages, and can predict the outbreak.

Hallucinations of the different senses are common in mania, and illusions still more so. In 178 out of 229 cases Brierre de Boismont professes to have met with such complications; they are generally fleeting, like the other morbid phenomena. Some have thought that the long endurance of the great expenditure of energy in acute mania is owing to a perversion of the muscular sense, by reason of which the true condition of the muscles is not declared. There can be little doubt that illusions of the muscular sense are at the root of the delusions with regard to bodily movements sometimes exhibited in mania; when a person lying in bed believes his limbs or himself to be flying through the air, it is certain that the muscular sense does not give correct information, but is affected with hallucinations.

The bodily functions often bear the great mental agitation of acute mania in a surprising manner. The pulse may be a little quicker in the early stage, when there is perhaps some febrile disturbance; but it is afterwards scarce raised in frequency. The temperature of the body is only slightly increased; but in cases of a typhoid type, where there is sleeplessness, restlessness, gradual wasting, and where the tendency is to death from exhaustion, Dr. Saunders has found it to be often raised from three to five degrees above the natural standard.[1]

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[1] Report of the Devon County Asylum for 1864.—Dr. Clouston has recently made some careful researches respecting the temperature of the insane. He finds the evening temperature of every form of insanity to be higher than the evening temperature of health, and excitement to be almost always attended by an increased temperature. (Journ. Ment. Science, April 1868.)

In the Insanity occurring after acute diseases, Dr. Weber's observations show only a slight increase of temperature, although this had been raised several degrees during the previous disease, and immediately rose again on the occasion of a relapse.[1] The skin may be either dry and harsh, or moist and of offensive odour. Constipation is common, but in some cases there occurs a continued and obstinate relaxation of the bowels. The urine Dr. Sutherland found to contain an excess of phosphates in acute mania; and if this were true, it would testify, like the increase of temperature, to an abnormal disintegration of tissue. More recent examinations of the urine, by Dr. Addison, result in the assertion that "the quantities of the urine, of the chloride of sodium, of the urea, phosphoric and sulphuric acids, excreted during the course of a maniacal paroxysm, occurring in acute mania, epilepsy, general paralysis, melancholia, or dementia, are less than the amounts excreted in an equal time during health."[2]

The *course* of mania is not often regularly progressive; there are generally remissions, and sometimes complete intermissions, or even so-called lucid intervals. The attacks may return at regular or irregular intervals, and thus constitute a periodic or recurrent mania; or attacks of melancholia may alternate with them, and give rise to what the French have described as *folie circulaire,* or *folie à double forme.* The duration of an attack of mania may be for hours or months, and recovery may be sudden or gradual. There can be no question of the occasional occurrence of a short maniacal fury, a *furor transitorius,* lasting for a few hours or days, usually associated with vivid hallucinations, and comparable to an attack of epilepsy,[3] and it is interesting to observe that these attacks are sometimes preceded by a strange anomalous sensation rising, like an epileptic aura, from some part of the body to the brain. When recovery takes place, it is usually within the year; it is rare after two years; and, indeed, the longer the disease lasts the worse is the prognosis, which is always unfavourable in recurrent mania and in mania alternating with melancholia. Recovery not taking place, the disease passes into chronic mania, or into dementia, or ends fatally. Death may be due to exhaustion, or to some intercurrent disease, such as pleurisy or pneumonia. When maniacal exhaustion proves fatal, the end may be very sudden and unexpected, so as to leave in the mind an anxious feeling of doubt whether a more energetic treatment might not have prevented death, or, if energetic treatment has been employed, whether that has not had something to do with hastening the fatal issue.

I have described the general features of the typical form of acute mania, but we meet with several varieties in practice. There is a form of very acute mania, which might properly be called an *acute delirious mania* or *acute maniacal delirium.* It is characterised by intense excitement, great restlessness and jactitation, entire incoherence, there not being the coherence of distinct and enduring delusion, and by only the briefest flash of momentary consciousness of what is going on around, or by apparent unconsciousness, except so far as fragments of impressions are caught up, whirled and lost in the agitation of the delirium. It runs a rapid course, very often to exhaustion and death, the pulse becoming quick and feeble, the skin hot, and the excitement and restlessness continuing to the last.

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[1] On the Delirium of Acute Insanity during the Decline of Acute Diseases; Med-Chir. Trans. vol. xlviii.

[2] On the Urine of the Insane, by A. Addison, M.D.; British and Foreign Med.-Chir. Review, 1865.

[3] Ueber Mania Transitoria, von Dr. L. Meyer. Virchow's Archiv, Band viii. Die Lehre von der Mania Transitoria, von Dr. R. Krafft-Ebing. Erlangen, 1865.

Puerperal Mania comes on within one month of parturition, and, like the Insanity of pregnancy, occurs most often in primiparæ. It is of an acute and incoherent character, marked by noisy restlessness, sleeplessness, tearing of clothes, hallucinations, and in some cases by great salacity, which is probably the direct mental effect of the irritation of the generative organs. Suicide may be attempted in an excited, purposeless way. The bodily symptoms, contradicting the violence of the mental excitement, indicate feebleness; the features are pinched, the skin is pale, cold, and clammy, and the pulse is quick, small, and irritable. Recovery takes place in three out of four cases of puerperal mania, usually in a few weeks; the patient, after the acute symptoms have subsided, sinking into a temporary state of confusion and feebleness of mind, and then waking up as from a dream.

Recurrent Mania reminds us of nothing so much as epilepsy in the regularity of its recurrence, in the uniformity of the symptoms of the attack, each being almost an exact image of the other, in its comparatively short duration, and in the temporary recovery. The patient becomes elated, hilarious, talkative, passing soon into a state of acute, noisy, and self-conscious mania, which may last for two or three weeks or longer, and then sinking into a brief stage of more or less depression or confusion of mind, whence he awakens to calmness and lucidity. In vain we flatter ourselves with the hope of recovery; after an interval of perfect lucidity, of varying duration in different cases, the attack recurs, goes through the same stages, and ends in the same way, only to be followed by other attacks, until at last the mind is weakened and there are no longer lucid intervals. Could we stop the attacks, mental power might still be regained by degrees, but we cannot; all the resources of our art fail to touch them, and I know no other form of Insanity which, while having so much the air of being curable, thus far defies all efforts to stay its course.

When the acute symptoms of mania have subsided, and the disease has become chronic, it presents most varied characters, according to its cause and the degree of mental degeneration. When there is considerable intellectual power apart from the delusions, as there usually is when the disease has been produced by moral causes, then the case may properly fall under monomania, or partial derangement of the faculties; when there is great loss of mental power together with delusions, as there often is when the disease has followed acute mania or a physical cause, then it may properly fall into one or other of the groups of dementia. On the one hand, then, chronic mania runs insensibly into monomania; on the other, into dementia. It is remarkable in some cases how much intellectual power may co-exist with extravagant delusions: a person who fancies that not an event in Europe happens which has not some hidden relation to him, who detects a plot against himself in the meeting of a cabinet, or in the journey of an emperor to his country palace, may yet have an exact knowledge of all his affairs, and be capable of giving a good opinion with regard to them. But what such a person cannot be depended on to do is to control his conduct.

The form of mental derangement produced by self-abuse—the *Insanity of masturbation*—furnishes a good example of a chronic mania in which there are no acute symptoms, the onset of the disease being most gradual. The patient becomes offensively egotistic; he is full of self-feeling and self-conceit; insensible to the claims of others upon him and of his duties to them; interested only in hypochondriacally watching his morbid sensations and feelings. His mental energy is sapped, and though he has extravagant pretensions, and often speaks of great projects engendered of his conceit, he never works systematically for any aim, but exhibits an incredible vacillation of conduct, and spends his days in indolent and self-suspicious self-brooding. His relatives he thinks hostile to him, because they do not show the interest in his sufferings which he craves, nor yield sufficiently to his pretensions. As matters get worse, the general suspicion of the hostility of people takes more definite form, and delusions spring up that persons speak offensively of him or watch him in the street, or comment upon what passes in his mind, or play tricks upon him by electricity or mesmerism, or in some other mysterious way. Still he professes the most exalted moral or religious aims. A later and worse stage is one of moody self-absorption and of extreme loss of mental power. He is silent, or if he converses he discovers delusions of a suspicious or obscene character, the perverted sexual passion still giving the colour to his thoughts. He dies at the last a miserable wreck. This is a form of insanity which has certainly its special exciting cause and its characteristic features; nevertheless, self-abuse seldom, if ever, produces it without the co-operation of the insane neurosis.

Monomania; Partial Mania; Partial Insanity; Delusional Insanity.—The exalted self-feeling which in acute mania uttered itself chiefly in turbulent action gets embodied in a fixed delusion, or in a group of delusions, which fails not still to testify the overweening self-esteem. As in melancholia the feeling of oppression of self was condensed into a delusion of being possessed with a devil, or otherwise afflicted, so here the exaggerated self-feeling is clothed in a corresponding delusion of power or grandeur, and the personality of the patient is transformed accordingly: he would fain have us believe that he has resolved the most abstruse problems of science; that he has devised an infallible scheme for reforming the world; that he is king, prophet, or divine. Monomania is, then, a partial ideational insanity, with overweening estimate of self, and fixed delusion or delusions upon one subject or a few subjects, apart from which the patient reasons tolerably correctly. Pathologically it represents a systematisation of the morbid action in the supreme cerebral centres—the establishment of a definite type of morbid nutrition in them. Having regard to the mode of origin of the delusion, the deep hold which the manner of its genesis proves it to have on the individual nature, it will be seen how erroneous it is to speak of the mind as sound apart from the delusion. As in melancholia so here, there is a fundamental affective disorder incapacitating the individual from a just appreciation of those things that really affect the self, that touch to the quick those genuine feelings revealing his innermost nature and instigating his conduct; and he is liable at any time to outbreaks of fury, which, like the delusion, are but expressions of the deep-rooted derangement. The mind is not unsound upon one point, but an unsound mind expresses itself in a particular morbid action. Patients thus suffering often seem calm and harmless enough under the regular discipline of an asylum; but if they are exposed to the excitement of ordinary life, seriously crossed in some project, or subjected to the stress of adverse events, they are liable to outbursts of uncontrollable rage or of true mania; so that one who may have been hitherto only interested by their harmless delusions, will be horrified at the utter madness which they exhibit.

The particular delusions of the monomaniac differ according to his occupation in life and the degree of his culture, and are frequently coloured by the events, social, religious, or political, of the epoch: Esquirol boasted that he could write the history of the French Revolution from the character of the Insanity which accompanied its different phases. Hallucinations and illusions frequently accompany the delusions which they appear sometimes to generate and always to strengthen. The behaviour of the patient accords with the character of his delusions: one makes sweeping plans and projects, enters upon vast speculations, and sometimes goes through an immense amount of patient and systematic work in perfecting some marvellous scientific invention; another reveals in gait and manner the exalted character of his delusion; and to a third, ordinary language does not suffice to express the magnificence of his ideas, and he invents new and mysterious signs, which, unintelligible to every one else, have wonderful significance for him.

The *course* of monomania, once established, is very seldom towards recovery; for as it is rarely primary, it represents a further degeneration or more advanced morbid action than mania or melancholia, upon which it usually follows. Even when it is primary, the outlook is not much more favourable, for it is then commonly secondary to some fundamental vice of character. Certainly recovery may take place, and the patient awake to sense as out of a dream; and in some rare instances it has taken place after years, especially under the influence of the revolution in the system produced by some intercurrent disease or at the climacteric period. When recovery does not occur, the disease becomes more chronic, lasting as such, or passes into actual dementia; the more the exaggerated self-feeling which inspires the delusion wanes, and the more this, losing its inspiration, becomes a mere form of words, the nearer the case gets towards incoherent dementia. As a general rule, it may be said that recovery does not take place when a fixed delusion has lasted for more than half a year.

Dementia.—It is the natural termination of mental degeneration, whether going on in the individual or through generations; and it is accordingly in the great majority of cases chronic, and secondary to some other form of mental disease. Dementia may, however, be both acute and primary, and is then not always distinguished from melancholia with stupor.

Acute dementia sometimes occurs after an attempt at strangulation, after certain acute diseases, and after a series of severe epileptic fits; and in one case under my observation a masked epilepsy seemed to take this form. As a primary disease, it sometimes follows a sudden and severe moral shock, and is now and then met with in young men and women, obscurely connected apparently with the state of the sexual functions. Dr. Skae describes a sexual or post-connubial mania taking the form of acute dementia, met with both in the male and female sex, but more frequently in the latter, and connected, he believes, with the effect produced on the nervous system by sexual intercourse. With these exceptions, dementia is a chronic and secondary disease presenting every possible variety in the degree of mental decay.

After the disappearance of a severe attack of acute mania, the effects of the shock are oftentimes visible in a certain condition of mental weakness without actual intellectual disorder: the force of character seems to have been sapped, and the finer moral and æsthetic feelings, which are, as it were, the bloom of culture, are abolished; the physiognomy has lost its highest expression, and the individual produces the impression of a certain childishness. This is one end of the scale of degeneration; but at the other extremity mental power is almost obliterated, the acquisitions of the past being completely razed out, and no interest in the present possible, and the patient leads a mere vegetative life. Between these two extremes every sort of transition is met with in practice, so that it is impossible here to do more than indicate certain prominent types.

Most of the permanent residents in asylums consist of those who, after mania, monomania, and melancholia, have subsided into a chronic state of more or less feebleness and incoherence of mind—the *crazy* people, who represent the wrecks of these forms of mental disease. Some there are who exhibit a few striking delusions which seem to be automatically expressed; the strong self-feeling which underlies or inspires these in monomania has faded away, and they are no longer full of self-assertion, nor eager, earnest, and consistent in carrying out their plans. The old paths of associations are broken up, and memory is almost abolished; all liveliness of feeling is gone, and there is little or no interest in what is going on around; and the only momentary excitement which occurs is when fixed delusions are attacked. It is remarkable, however, how even in these cases the excitement of a fever will sometimes restore temporarily the functions of the mind which seemed to have gone for ever. The countenance no longer expresses any fixed passion; there is a want of harmony, or, as it were, a dislocation of its features, and the most which it manifests is the shivered expression of a passion or the shattered wreck of a smile. There is a corresponding imbecility on the motor side: some can certainly continue their former occupation, or can do a little simple manual work, but there is no systematic correspondence of action to delusions, and there is not unfrequently a useless and busy industry in gathering stones, pieces of paper, and sticks. Strange propensities of all kinds are exhibited, as for example to stand or crouch in a particular corner, to walk backwards and forwards for a certain distance on a particular slip of ground, or to fantastically ornament the person with feathers or flowers. The mood may be of surly depression, or of more or less exaltation. Hallucinations and illusions of the extremest kind are frequent, and tend to sustain the delusions: one woman has in her belly the whole tribe of the apostles, prophets, and martyrs; another lovingly nurses as her child a lump of wood decked in rags; a third person, whose singular movements seem unaccountable, is busy spinning threads out of sunbeams; while a fourth continues the most violent movements of his arms in order to prevent the motion of the universe or of his own blood from coming to a stand. The bodily health is usually good, the patient frequently getting stout as the active symptoms of mania or melancholia subside into the calm of dementia. This group represents for the most part dementia following on monomania.

In another group of cases there is greater external activity, with a more general incoherence or craziness. There are no fixed delusions, although there is evidence in the patient's incoherent expressions, or in his senseless, parrot-like repetition of certain words, of the wreck of such as existed in the maniacal stage. The senses are open to the reception of impressions, but these do not seem to be further fashioned into ideas. There is sometimes entire indifference to surroundings, together with great insensibility to pain; or there are short outbreaks of incoherent passion or fury; or there may be desperate and unaccountable homicidal violence. The predominant mood is different in different cases: some are gay, happy, and prone to laugh and chatter; others are gloomy, weep, and display the mimicry of sorrow; while others again are malicious, spiteful, and addicted to a purposeless mischief with a monkey-like cunning and persistence. The loss of memory is marked: some have entirely forgotten their former life and their own names, while others, who perhaps forget instantly the last thing said, can reproduce the distant past with fidelity. In the movements of some there is marked feebleness, or the indication of commencing paralysis; while others are restless, agitated, and run about with ceaseless activity. The bodily health is usually good: they sleep well, and eat well, often gluttonously and without discrimination, and are sometimes prone to get fat until an attack of excitement and agitation, to which some are periodically liable, reduces them. The physiognomy is blank and expressionless, especially so when the patient is addressed; it is also prematurely aged.

Lastly, there is a group of demented patients, in whom the mind is almost extinguished; who must be fed, moved, clothed, and cared for; who manifest little or no sensibility; whose only utterance is a grunt or a cry; and whose only movements are to rub their heads or hands. Their existence is little more than organic, and if not carried off by pneumonia, tubercle, or some other disease, as they frequently are, they die from effusion on the brain, serous or hæmorrhagic, or from atrophy of the brain, or from the effects of accident, to which, through their apathetic helplessness, they are much exposed.

Senile dementia is characterised by weakness of mind, inability to grasp the present, and great loss of memory, especially of recent events; the patient talking of events long past as if they had just happened, perhaps believing himself to be in daily intercourse with persons who have been long dead, and confounding his present life, the events of which are almost immediately forgotten, with his past life.

The *course* of secondary dementia is from bad to worse: it is impossible that recovery should take place, although the condition and habits of a patient may be much improved by proper care. Those who suffer from acute primary dementia get well generally; but, of course, senile dementia, though primary, is beyond the reach of remedy. Death may be due to effusion on the brain, or to atrophy of it, or it is produced by accidental disease, as tubercle or pneumonia. Dementia is the only form of Insanity in which the average temperature is below that of health.

Moral Insanity.—Under this unfortunate name, Dr. Prichard described cases of real mental disorder in which, without hallucination, illusion, or delusion, the derangement is exhibited in a perverted state of those mental faculties which are called the active and moral powers—the feelings, affections, propensities, and conduct. He never meant that a vicious act or crime, however extreme, should be deemed proof of moral insanity; for he expressly insists on tracing the disorder to certain recognisable causes of disease. "There is often," he says, "a strong hereditary tendency to Insanity; the individual has previously suffered from an attack of madness of a decided character; there has been some great moral shock, as a loss of fortune; or there has been some severe physical shock, as an attack of paralysis or epilepsy, or some febrile or inflammatory disorder, which has produced a perceptible change in the habitual state of the constitution. In all these cases, there has been an alteration in the temper and habits."[1] In reality, however, this moral insanity is no special form of disease, but a variety of that mental derangement already described as *affective* or *pathetic;* and briefly to enumerate the varieties of this affective form of derangement, all which were confounded by Pinel under *mania sine delirio,* will best exhibit the nature and relations of moral insanity.

(*a*) There are attacks of derangement in which the moral or affective alienation is very great, and in which the intellect is only secondarily affected through the moral perversion, the patient reasoning very well from the premises of his perverted feelings; he has no delusion unless his whole manner of thought in reference to self be called a delusion. These attacks are often associated with epilepsy, which they may immediately precede, as they sometimes precede an outbreak of mania; or they may occur at periodical intervals for months before actual epilepsy, and sometimes take the place of the true epileptic seizure afterwards; or again, the epileptic fits may cease entirely, and be followed by such attacks of profound moral derangement, occurring at uncertain periods, and perhaps passing into dementia. It is important to bear in mind that when associated with the epileptic neurosis they represent a condition in which vivid hallucinations and irresistible impulses of a desperate kind, homicidal or suicidal, are apt to arise instantaneously; that they, in truth, embrace the most dangerous forms of the so-called impulsive insanity—the *monomanie instinctive* of Esquirol.

(*b*) There is the melancholic depression of the affective life already described—simple melancholia, in which the anguish rises to such a pitch as to issue in an explosion of convulsive violence, homicidal or suicidal, no fixed delusion being present. Some of the cases of so-called impulsive insanity are examples of this form of disease.

(*c*) The moral insanity proper of Prichard (*monomanie raisonnante,* Esquirol) occurs in most instances as the result of hereditary taint, aggravated or not by unfavourable conditions of life. It is a more advanced stage of degeneration than that which has been described as the insane temperament, but it does not reach actual intellectual derangement: the moral feeling being the highest acquisition of human culture in the course of development through the ages, its loss is one of the earliest effects of degeneration. Moreover, it will always be necessary to consider the social condition of any one suspected to have moral insanity, inasmuch as it is in the loss of the social feeling by reason of disease that the alienation essentially consists. If a person in a good position, possessed of the feelings that belong to a certain social state, does, after a cause known to be capable of producing every kind of Insanity, undergo a great change of character, lose all good feelings, and from being truthful, temperate, and considerate, become a shameless liar, shamelessly vicious and brutally perverse, then it is impossible not to see the effects of disease. Friends and relatives may remonstrate with such a one, and punishment may be allowed to have its due course; but in the end both friends and every one who has to do with him must confess that he is the victim of disease—that his proper place is not the prison, but the asylum. Such moral alienation may occur after previous attacks of Insanity, after acute fevers, after some form of brain disease, or after injury to the head. After an attack or two of melancholia with suicidal tendency, from which recovery has taken place, the patient is perhaps attacked with genuine moral insanity, which ultimately passes into intellectual disorder and dementia. Or there has been more or less congenital moral defect, and maniacal exacerbations of actual moral insanity, without positive intellectual disorder, take place, dementia following after a time; these outbreaks may occur at puberty, or at the menstrual periods. A moral insanity again is in some instances the first stage of mental degeneration that is produced by self-abuse or sexual excesses: it now and then occurs in consequence of a severe moral shock, as the forerunner of more marked Insanity; and it not unfrequently precedes general paralysis. If the evidence from its own nature and causation were insufficient, the simple fact that it is often the forerunner of the severest mental disease, might suffice to teach the right interpretation of moral insanity.

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[1] A Treatise on Insanity and other Disorders affecting the Mind. By J. C. Prichard, M.D. F.R.S

(*d*) There are certain beings who are truly moral imbeciles, the original defect being due, as in idiocy, to some cause acting either before birth, or during the first years of life. With such moral defect there is often associated more or less intellectual imbecility, though not always plainly so; it is remarkable indeed what an acute intellect may sometimes co-exist with an entire absence of the moral sense. Some of the notorious gaol-birds amongst the criminal classes belong to this group; and in higher social spheres there are now and then met with unhappy creatures who, from their earliest years, have been addicted to lying, or stealing, or every sort of vicious act—who have been expelled from school after school, the hopeless pupils of many masters, and who finally end in an asylum. They are instinctively vicious or criminal, exhibit a complete moral insensibility, and commonly masturbate; and they sometimes decline into mania and dementia.

Here, then, may conveniently be summed up in groups, according to their most prominent symptoms, the various forms of Insanity described, idiocy and general paralysis, which yet remain to be described, being added :—

I.—AFFECTIVE OR PATHETIC INSANITY.

1. *Maniacal perversion of the affective life. Mania sine delirio.*
2. *Melancholic depression, or Simple Melancholia.*
3. *Moral alienation proper;* approaching which, but not reaching the degree of positive insanity, is *the insane temperament, or the neurosis spasmodica.*

II. IDEATIONAL INSANITY.

1. *General:*
    - a. *Mania*
        - *Acute.*
        - *Chronic.*
        - *Recurrent.*
    - b. *Acute Melancholia.*
2. *Partial:*
    - a. *Monomania.*
    - b. *Melancholia. Lypemania.*
3. *Dementia*
    - *Primary.*
    - *Secondary.*
4. *Idiocy.*
5. *General Paralysis.*

In making use of this or any other provisional classification of symptoms, it should be clearly understood that the forms of Insanity are not actual pathological entities, but different degrees or kinds of the degeneration of the mental organization—in other words, of deviation from healthy mental life: and they are consequently sometimes found intermixed, replacing one another, or manifested in successive order in the same individual. There is a strong propensity not only to make divisions in knowledge where there are none in nature, and then to impose the divisions upon nature, making the reality thus conformable to the idea, but to go farther than that and to convert the generalisations from observation into positive entities, and then to permit these creations to tyrannize over the thoughts. A typical case of madness might be described as one in which the disorder, commencing in emotional disturbance and eccentricities of actions—in derangement of the affective life, passes thence into melancholia or mania, and finally, by a further declension, into dementia. This also is the natural course of mental degeneration proceeding unchecked through generations. The necessity of describing different forms of Insanity under different names should never lead to a neglect of the real relations which they have to one another as different stages of deviation from that mental life which we agree to regard as ideal or typical.

Idiocy.—It is an arrest of mental development by reason of some defect of cerebral development which is either congenital or occurs soon after birth. It will not appear strange that such defect is not always detectable when we reflect that the development of the brain as the ministering organ of the mental life really takes place after birth, and that an arrest thereof would take place within the recesses of the intimate activity of nervous element to which our senses have not gained access—where the subtlety of nature yet far exceeds the subtlety of human research. Marked imperfections of the brain are undoubtedly often met with in idiots. It is sometimes abnormally small, the general arrest of growth being due either to some condition of defective bodily nutrition, or to a premature ossification of the sutures of the skull and a consequent prevention of the growth which normally takes place actively during the first years of life. While it may be that there is no other defect than the abnormal smallness, it happens much more frequently that there are other anomalies, as hydrocephalus, unequal size of the hemispheres, and deficient development of the convolutions. All degrees of unequal size of the hemispheres have been met with, from that slight difference which is natural, to that extreme degree where a whole hemisphere has been replaced by a meshwork filled with fluid. Again, there is scarce a particular part of the brain which has not occasionally been found wanting: the corpus callosum may be defective or absent; there may be a deficient development of the anterior lobes, and a simplicity of the convolutions, such as belong to the lower animals; or the posterior lobes may not extend far enough back to cover the cerebellum, as normally they do not in some monkeys, and in all the animals below them. Chronic hydrocephalus, apparently primary in some cases, but in many others secondary to the atrophy or defect of brain, is frequent in idiots, and sometimes makes them large-headed: the serous fluid may exist in large quantity within the ventricles without being fatal, and death ultimately occur suddenly from a slight increase of it.[1] Sclerosis of the brain-substance often accompanies atrophy, or defect of development.

The irregularities of the skull in idiocy have been much studied of late. When the development of the brain is simply arrested, the growth of the bone may be arrested also, and then the skull is *microcephalic*. More often it would appear that owing to some constitutional defect of nutrition the arrest of the growth of the bone is primary, and a premature closure of the sutures takes place, whence follows a narrowing or shortening of the skull. Compensating enlargements thereupon take place in some cases, the growth of the brain being in the direction of least resistance, and increasing the cranial deformity though making the mischief less. According to the suture prematurely closed, and to the amount and character of the compensating enlargement, will be the degree and kind of the deformity, of which many kinds have been described. Virchow has investigated with great care what he calls the tribasilar synostosis, which, occurring at the base of the skull, is the anatomical condition of the skull of Cretinism.

The causes of idiocy are sometimes traceable to parents. Frequent intermarriages in families lead to a degeneration that manifests itself in deaf mutism, albinoism, and idiocy; parental intemperance and excess, according to Dr. Howe,[2] occupy a high place as causes; and the natural term of Insanity proceeding unchecked through generations is, as Morel[1] has shown, sterile idiocy. During fœtal life great fright or mental agitation in the mother or irregularities and excesses on her part may lead to mental defect in the child. But perhaps the most frequent causes of an arrest of mental development are those which operate after birth up to the third or fourth year: they are epilepsy, the acute exanthemata, perhaps syphilis, and certainly conditions of bad nutrition, such as are produced by overcrowding, dirt, and want. Cretinism is an endemic idiocy arising out of unknown territorial conditions.

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[1] On Serous Effusion from the Membranes and into the Ventricles of the Brain, by John Sims, M.D.; Med.-Chir. Trans. vol. xix.—Clinical Notes on Chronic Hydrocephalus in the Adult, by S. Wilks, M.D.; Journ. Ment. Science, January 1865.

[2] Report on the Causes of Idiocy.

The extremest idiots are destitute of any intelligence whatever; they are apathetic, torpid beings, having a human semblance, whom it is necessary to feed, to move, to clothe, to take care of in every particular; who can attend to nothing, and remember nothing; who cannot speak a word; who grunt, make unintelligible sounds, and are unquiet if their appetites are not satisfied, or mechanically continue some automatic movement of hand, head, or body. The senses are almost invariably defective or wanting, the sensibility of the skin being commonly very imperfect, the hearing feeble, and smell and taste so deficient or perverted that the most acrid or filthy matters are eaten with indifference. The muscular development shares in the general defect; there are cramps of the limbs, contractions or paralysis of certain muscles, and epileptic convulsions. In Scotland there were, five years ago, 2,236 imbeciles and idiots, of whom 43 were paralytic, 46 hemiplegic, 10 paraplegic, 17 choreic, and not less than 207 epileptic.[2] In less extreme cases there is evident want of power over the muscles; the walk is staggering and uncertain, the eye rolls vacantly, strabismus is common, the speech is defective, and there is slavering at the mouth. Sexual power is absent in the worst cases, and notwithstanding the self-abuse practised by some idiots, feebleness is in all cases more common than excess.

At the other end of the scale stand the so-called imbeciles, the highest of whom are only a little lower than those simple-minded people not deemed imbecile. The difference again between imbecility and idiocy is only one of degree, so that it is impossible to define it. In all sorts and conditions of idiocy two principal types may be broadly distinguished: one embracing the torpid and apathetic beings, who have usually some bodily deformity, and who give feeble signs of life; the other, those lively and excited beings who, rarely observably deformed, are unquiet and restlessly respondent to a rapid succession of impressions, who shriek, laugh, weep, gesticulate, clap their hands, get into mischief, and sometimes pass by a turbulent declension into true maniacal fury. Even the quiet idiots are occasionally liable to sudden attacks of fury, in which they bite, shriek, scratch, kick, beat their heads against the walls, and in other ways act viciously enough. Special talents or aptitudes of a remarkable kind, for remembering, for drawing, or for music, which seem quite inconsistent with the general character of their intelligence, are sometimes exhibited by idiots whose disease is of hereditary origin.

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[1] Traité des Degénérescences physiques, intellectuelles et morales, de l'Espèce humaine, par Dr. B. A. Morel, 1857.

[2] The Psychology of Idiocy; Journ. Ment. Science, April 1865.—Epileptics: their Mental Condition, by W. A. F. Browne, Commissioner in Lunacy for Scotland; Journ. Ment. Science, October 1865.

Esquirol divided idiots into three classes, according to the condition of speech. The first division included those who could use words and short phrases; the second, those who could only utter monosyllables and certain cries; the third, those who had neither speech nor monosyllable. Dr. Hack Tuke[1] proposes a physiological division of them into, first, those who exhibit only reflex or excitomotor movements; secondly, those whose acts are sensorimotor and ideomotor; and thirdly, those who manifest volition. It is a division which, not perhaps practically available, serves to mark the different degrees of degeneracy. By Griesinger, idiots are grouped in certain types:—1. Well-formed children in whom the mental development, which remains at the lowest grade, is the only apparent defect, the defect not being due to any hereditary, but to some accidental cause of degeneration. 2. The cases in which both bodily and mental development have been palpably arrested: these are the dwarfs in mind and body. 3. The Cretin, or basilar-synostotic type of idiocy or imbecility. Cretinism generally manifests itself a few months after birth, and is frequently associated with bodily deformity and goitre; and it is supposed to be due to some miasmatic influence primarily affecting the growth of the bones of the skull. It is most common amongst the mountains of Switzerland, but is met with sometimes in badly-drained places, and now and then sporadically. 4. The Aztec type, consisting of the true microcephalic idiots. 5. The theroid idiots, who have a sort of resemblance to some animal. Pinel, for example, gives a striking account of an idiot who was very like a sheep in habits and manner; and some idiots irresistibly bring to mind the monkey. Still, however degraded an idiot may be, he never really reverts to an animal type; for he represents a new and morbid variety, which, but for the fostering care of higher beings, would speedily be extinguished. ✗ [illegible]

Though idiots can never reach a normal development, their condition in many cases may be much improved by persevering training. The faculties which they do possess may thus be brought out in a remarkable manner, and they be made automatically skilful in certain tasks. When epilepsy has co-existed with the idiocy, and afterwards disappeared, marked improvements may take place. Idiots very seldom attain old age; they are "old in their youth, and die ere middle age," apparently from lack of vitality. In some cases, the disease of brain—atrophy or hydrocephalus—directly leads to death.

General Paralysis.—It is a form of Insanity, first described by Bayle and Calmeil, which is characterised by a progressive diminution of mental power, and by a paralysis which gradually increases and invades the whole muscular system. It is far more frequent amongst men than women, and its most frequent cause is intemperance of some kind. Two of the best marked examples of this disease which I have seen, occurred, however, in teetotallers, who never had been given to alcoholic excess; but in both there was hereditary taint, and in both there was reason to suspect enervating, though marital, sexual excess.

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[1] Manual of Psychological Medicine, by Dr. Bucknill and Dr. D. Hack Tuke.

Much discussion has taken place as to whether the mental symptoms precede the paralytic phenomena, or whether the latter first appear—whether the Insanity is primary, or whether, as Baillarger holds, the paralysis is the primary and main affection, the Insanity secondary and accessory. There can be no question in the minds of those who, unbiassed by any theory, simply observe cases, that the mental disorder does sometimes appear simultaneously with the motor disorder; that far more frequently, indeed most frequently of all, the mental symptoms are observed some time before there is any trace of paralysis; but that in some few cases the paralytic phenomena do precede by a short period the mental symptoms. In fifty-one cases out of eighty-six observed carefully by Parchappe, he found the paralysis and mental disorder to be simultaneous; in twenty-seven cases the paralysis was subsequent, and in eight the precedence was undetermined. Leidesdorf has related one case in which the earliest symptoms were spinal; and one or two similar cases have been recorded.[1]

The motor symptoms are first witnessed in the tongue, which has to execute the most delicate and complex movements with so much precision, and especially in the articulation of words abounding in consonants, where the most complex co-ordination is required. When the patient speaks earnestly, he does not articulate exactly, and there is a certain pause or indecision detectable in his utterance, as if there was some difficulty in bringing out the word; in some cases the speech is slower, more deliberate, with a strong accentuation of and a lingering on the syllables, as if the patient were speaking with great consideration. When the tongue is put out, which is done with some difficulty, there is a fibrillar quivering or trembling of its muscles, but it is not pulled to one side. There is a tremulousness, also, in the muscles of expression when put in action—especially in those of the lips, which quiver as in one just about to burst into tears. These phenomena, which are of fatal omen, may not be apparent at first, when the patient is calm and collected; but if he has had a sleepless night, or if he is much excited from any cause, then they become evident. An inequality in the size of the pupils is often an early symptom, but it is not a characteristic one; it is sometimes present in other forms of Insanity, and it is not always present in general paralysis. A transitory squint is observed occasionally at the commencement of the disease, and at a later period a slight ptosis of the upper eyelid. As the disease increases, the patient's walk becomes affected : the feet are not properly raised and firmly put down on the ground; he easily stumbles at a step or on uneven ground, and if asked suddenly to turn round when walking straight forward, he will stagger like a drunken man. He may nevertheless be very active in walking, and commonly thinks himself wonderfully well and strong. Precise co-ordination of movement, such as is necessary for writing, sewing, and like acquired automatic acts, is lost. The disease still advancing, the articulation becomes less distinct, the walk more and more tottering; the knees fail; the patient frequently tumbles, and finally cannot get up again without help. At last the primary automatic or reflex movements fail; the pupils become dilated, but unequal in size; the sphincters lose their power, and the patient may be choked by a lump of food getting into his larynx. Sometimes there are transitory contractions of arm or leg, and a grinding or gnashing of the teeth is not uncommon. The contractility of muscles to the electric stimulus is retained.

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[1] Beiträge zur Diagnostik der Geisteskrankheiten. Von Dr. M. Leidesdorf.

Cutaneous sensibility is usually diminished in the early stages, and at a later period it is sometimes lost. Yet there are occasionally transitory conditions of extreme hyperæsthesia, so that the patient shrieks out in great agony, or the slightest touch produces reflex movements or even convulsions. The muscular sense is especially affected, so that the sufferer, deprived of power of executing all complex and delicate movements, deems himself not less skilful than when at his best state, or, quite paralysed, believes himself to have a giant's strength. The special senses are commonly unaffected until near the end, when smell and taste are diminished or lost, and vision fails. Sometimes, however, the patient has vivid hallucinations in the night : there were glorious visions of angels descending from heaven on ladders of gold in one patient under my care, and an agonizing vision of his own wife in the act of adultery rendered another frantic for a time. A great increase of sexual desire and an excited display of it are not unfrequent at the beginning of the disease, but there is not corresponding power; and what power there may be is soon quite lost.

The mental derangement is commonly marked by an exaggerated feeling of personal power and importance. After a brief stage of melancholic depression there is a notable change in the character, manifest in different ways : in one there is great mental excitement, and he is joyously and actively busy with wide-sweeping projects and speculations, indifferent to stern realities, and in all ways eager and ready to accomplish the impossible; in another there is a lack of former energy, and he is painfully troubled about little things, dull and confused in his thoughts, and demented in behaviour; while another exhibits unwonted perversities of feeling and conduct, such as mightily astonish his friends; he breaks out into sexual excesses quite foreign to his usual sober character, or orders numerous articles of jewellery for which he can never pay, or even steals what strikes his fancy. Begin as it may, the mental disorder, when unchecked, generally issues in incoherence and extravagant delusions as to personal power and grandeur : the miserable sufferer who can scarce support his tottering body avers that he has the might and vigour of Hercules; while industriously hoarding up pieces of rag, paper, or glass as articles of value, he will sign a cheque for countless millions, or make an easy present of New York; maintaining that he can command a king to do his pleasure, in the same breath he prays piteously to be allowed to go to his own humble home; or, with sexual power extinct, boasts exultantly that a princess shall be his wife and princes be born of his loins. An extreme loss of memory is in striking contrast with the semblance of exaltation : the patient forgets entirely how long he has been in confinement, or denies angrily that he has a wife, though recognising her gladly when she visits him. In some cases the delusions are of a terrific character and accompanied with great emotional depression ; and a day of melancholic depression may now and then intervene in the course of the exalted form. There is a class of patients who present in physiognomy and habit of body a mixture of stupidity and the deepest depression, and exhibit sad delusions of as extreme a character as the delusions of grandeur : they think themselves bodily transformed in whole or in part; that their body has been immensely enlarged, that their eyes cannot see, their ears cannot hear; that their throat is sealed up. Dr. Clouston has pointed out that there is an intimate relationship between this form of general paralysis with depressions and tuberculosis. In another variety, of rare occurrence, the mental disorder consists in a regular decline of intelligence—a gradually increasing stupidity from the first. Attacks of great excitement and blind violence frequently occur during the progress of the disease. During them L. Meyer has observed the temperature of the head to be raised, and after them the mental decay is found to have increased. As the disease approaches its end—the end of life—the dementia is extreme, and the face becomes an expressionless mask across which now and then flickers the broken ripple of a smile, or it is fixed in a ghastly, sardonic grin ; but even in the last stage of mental disorganization, when the capability of a distinct delusion is gone, the muttered words are oftentimes about golden carriages and millions of money.

The *course* of general paralysis is towards death, though not steadily so. Under proper treatment a great improvement takes place in the early stages, and the disease seems to be arrested. Some have thought that actual recovery does now and then take place ; and certainly there have been in exceptional cases intermissions of such a length that the disease has lasted for ten years. On the whole, however, it is irregularly progressive, its duration being usually from a few months to about three years. It hardly ever occurs before the age of thirty. In the more advanced stages sudden attacks of loss of consciousness, with epileptiform convulsions, are not unfrequent, after which the paralysis and mental decay are both found to have increased. Dr. Saunders has observed that the temperature of the body in general paralysis is generally one or two degrees below the average, but that it rises during the excesses of maniacal excitement, falling again as calmness returns. During the so-called congestive attacks, again, where there is complete coma or epileptiform convulsion, there is generally a considerable rise of temperature: in one case the temperature was for some time 98°, but it rose an hour after one of these attacks to 105°, and next day to 106°, the patient dying in thirty-six hours from the commencement of the attack.[1] Dr. Clouston has found that in general paralysis there may be a difference of 5·8° in the same individual in different stages of the disease. In the last miserable stage of all, when life flickers before extinction, large sloughing bedsores form, notwithstanding the best care, and diarrhœa or pneumonia hastens the long-expected ending.

DIAGNOSIS.—The diagnosis of Insanity is as difficult in some cases as it is easy in others. Acute mania cannot well be confounded with any other disease, and the only doubtful question in regard to it will be in the event of an impostor attempting to simulate it. He must be a clever impostor, however, who can simulate the wild restless eye, the quick fragmentary associations of ideas, the rapid capricious movements, and the volubility of utterance of acute mania, so as to deceive an experienced observer; nor can he, however skilful, pass days without sleep, and even weeks with only a few hours' sleep, maintaining a constant activity the while, as the maniac does. The skin in acute mania is dry and harsh, or clammy, but the skin of a pretender who tries to keep up the muscular agitation will hardly fail to be hot and sweating. *Meningitis* will be known from mania by its own positive symptoms—by the premonitory rigors, when they occur, the cephalalgia, the fever, the contracted pupil, and the intolerance of light; by a muscular activity, paroxysmal, not continuous, and by frequent spasms or convulsions; by the acute severity of the delirium and the vivid illusions of the senses; and by its rapid progress either to recovery or death. *Delirium tremens* will also be distinguished by its characteristic symptoms—the muscular tremors, the peculiar fearful illusions and hallucinations, the cold skin, feeble pulse, and white and tremulous tongue. It must not be forgotten, however, that persons who have a strong predisposition to Insanity, or who have been insane, or who have suffered at some time from a severe injury to the head, do sometimes after an alcoholic debauch become truly maniacal for a time. In this condition, which may be of short duration, dangerous hallucinations sometimes arise, and the sufferer may perpetrate crime, not knowing afterwards what he has done: of this fact a searching investigation of instances can leave no doubt. The result again of continued intemperance, or of a long debauch, is sometimes to produce, not a delirium tremens, but a true mania, marked by active and violent delirium.

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[1] Report of the Devon County Asylum for 1864.

Chronic mania is the most likely form of Insanity to be feigned, and if feigned with the skill of Hamlet, the very elect may be deceived. A vulgar impostor will, however, generally "o'erstep the modesty of nature," and overact the part in the extravagance of what he says, and in the absurdity or violence of what he does, while he will almost of a certainty fall short of his part in the emotional expression of the maniacal countenance. Having the popular notion that a lunatic is widely different from a sane person, he will entirely fail to understand the character which he wishes to represent, so that an experienced person may detect his ignorance; and believing that he must make a great display in order to produce an adequate impression, he will, like a bad actor, exaggerate and rant, so that any one with insight, though without special experience, may discover his incompetency. He will pretend that he cannot remember the simplest things, that he cannot reckon correctly, and will act extravagantly, and answer stupidly or wrongly, when a real lunatic would act calmly and answer intelligently or rightly; he will moreover show no irritation or anger at the suspicion of his sanity. It may not be amiss to suggest incidentally in his hearing some symptoms which he ought to exhibit, and to take notice whether he subsequently adopts the hint. If he refuses to converse, feigning apathetic dementia with dirty habits, which is perhaps the form of Insanity most often feigned, the diagnosis becomes more difficult, and a long observation may be necessary in order to establish it. It is truly astonishing how long an impostor will sometimes persist: one man, whose story Dr. Bucknill tells, kept up the pretence of Insanity for more than two years, and then broke down in his part. The two important considerations to be kept in view are, first, the existence of any motive for feigning, and, secondly, the consistency or inconsistency of the symptoms with a definite form of mental disease. Perhaps it would not be far from the truth to say that he who can feign madness so completely as to deceive an experienced observer, is not far from being the character which he represents; for unless there be real madness beneath the feigned phenomena, there will be some want of coherence in them as a whole, and an incongruity with any recognised form of mental disease.

The discovery of chronic mania or monomania where it does exist, but where the patient is suspicious and strives to hide it, may be a very difficult task. There is generally some sign of the disease in the countenance and bearing of the patient: "The principal characteristic in some is," says Dr. Bucknill, "a peculiar want of harmony in the expression of the features; in others, the fixed expression of some intense emotion is remarkable—of defiant pride, of sullen obstinacy, of smirking vanity, or of leering sensuality." The demeanour may be defiant, sullen, restless, or absorbed, and the clothing untidy and neglected: in both demeanour and dress there are sometimes peculiarities which, when bottomed, open up a secret mine of madness. To detect any delusion the patient should be examined carefully on all matters intimately touching himself, anything peculiar or notable in his expressions, or any obscure references, being watched for, noted in the mind, and subsequently quietly followed up. If he seems to pass hastily over, or to avoid, some subject, he should be unobtrusively but steadily pressed upon it; and if he declines to speak of the matter, or gets angry, the refusal or irritation is alike significant. All this should be done in as quiet and amiable a manner as possible, so as to avoid giving unnecessary offence, and to make it a difficult matter for the patient to take offence and break off the interview. It is an intellectual contest between a sound and an unsound intellect, in which the weakness of the latter is compensated by its acting on the defensive, the superiority of the former lessened by its acting on the offensive. Heinroth has affirmed, what is popularly thought, that though the insane may often *conceal* their delusions, they cannot *deny* them. This is by no means true of all cases: some will deny their delusions with as much energy as Peter showed when he denied the dangerous truth, or will even labour to explain them away as jokes. When all else fails, it may be worth while grievously to offend the patient's self-love, and to make him extremely angry, and in the fury of passion he will sometimes, notwithstanding his suspicion, reveal the hidden delusion. Failure will be rare with the expert who likes to be persistent and patient enough. In doubtful cases, it will always be well to get the patient to write, for it is truly surprising what extravagant delusions may be exhibited in a letter by one who manages to conceal them in conversation. Careful research should of course be made into the previous history, in order to discover whether there is hereditary taint, and what degree of it; whether there has been any previous attack of Insanity, and whether there has been any observed change of feelings, character, and habits, especially after some efficient cause of Insanity. At the outset a patient sometimes has a suspicion that he may be thought mad, and is very earnest and vehement in accounting for his morbid feelings, and at great pains to convince those around him that he is not mad.[1]

Melancholia is not usually difficult to recognise, as patients in most cases do not care to conceal their painful delusions; still there are some who not only conceal but deny them. A patient afflicted with homicidal and suicidal impulse, and intensely miserable in consequence, will positively conceal and deny the morbid impulse, in order to throw those around off their guard, and the better to effect his purpose; and more than one such, foolishly removed from under control by ignorant but well-meaning friends, has afterwards committed suicide or homicide, or both. Another, who cannot entirely conceal his disease, will even attribute his depression to the confinement which he is undergoing, and asseverate most earnestly that he will be quite well at home; this intense eagerness to be delivered from control being truly the surest sign that he is not fit to be from under control. In all such cases it is necessary to watch patiently from day to day; for it will sometimes happen that a delusion, denied on one day, is predominant on another, and it is very apt to become so, if the patient does not see a prospect of release through his simulation of sanity.

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[1] There are some excellent observations by Dr. Bucknill in the Manual of Psychological Medicine, on the mode of conducting the examination of an insane person, p. 310.

It may be important to distinguish between the hypochondriac and the melancholic, as the former, committing a murder, would certainly be hanged, the latter probably not. The hypochondriac refers his sufferings to some organic disturbance or disease, in which there may be more or less reality; he displays an exaggerated sensibility in regard to all his organic processes, or to some one of them in particular, so that he has either many delusions respecting his health, or his whole habit of thought respecting it is perverted; he is fond of talking of his sufferings, and of consulting medical men; he evinces a great love of life, and no disposition to commit suicide; his intellect is sound, and his feelings are not perverted, apart from questions touching his health. The melancholic, on the other hand, refers his sufferings to some groundless extraneous cause, either operating from without, or having taken possession of body or soul, or both, so that he has frequently a single and fixed delusion; his anguish is a mental anguish, and he asserts that medicine can do him no good; he is often suicidal; his affective life is profoundly implicated, and he is incapacitated from intellectual activity, though there may be no marked intellectual derangement apart from the delusion. It must not be overlooked, however, that hypochondriasis may pass into true melancholia, as well as co-exist with it, and that a true hypochondriacal melancholy may rise to such a pitch as to render the individual irresponsible for his actions.

Melancholia with stupor may easily be confounded with acute dementia; nor is it always possible to distinguish them at the outset. The expression of the melancholic is that of one astonied, or as if fixed in a painful trance—the mind veiled, as it were, by a great cloud let down between it and the external world. The patient stands or sits in one place, or moves slowly to and fro; he often offers a passive resistance to being moved, or to being fed; sometimes he exhibits a strong tendency to suicide, and, now and then, a temporary excitement; on recovery, he remembers his suffering as a painful dream, or as a strange and fearful trance, during which he was partly conscious of things around, but unable to express himself. In dementia, the countenance is expressionless; there is no resistance to being moved, or to food; the patient is not suicidal; the bodily functions are less affected; on recovery, there is no memory of the attack.

In certain criminal or civil trials, it may be necessary to distinguish between eccentricity and Insanity. There is a great gap between them: the truly eccentric man has a strong individuality, but little vanity; he has broad and original views, and great moral courage; he is emancipated from vulgar prejudice, and heeds not much the world's blame or censure; he differs from the majority perhaps because he is in advance of the habits and superstitions to which it is in bondage;

and he is not at all likely to become insane. But there is a weak affectation of eccentricity which is very apt to end in Insanity: with it are infected certain feeble-minded beings, often badly bred or badly trained, who are empty of any true individuality, but inflated with an excessive vanity; who have a small intellect which they use in the service of their passions; who do silly and eccentric things, not unconsciously as the spontaneous expression of their nature, but out of a morbid craving to attract attention; who represent a condition of mental derangement that is the forerunner of Insanity; who, when they are not given up to sexual excesses, are often masturbators.

In the diagnosis of so-called moral insanity, it is necessary to look for a sufficient cause of mental disease from which the vicious or violent acts may be logically traced through a train of symptoms, such as marked change of character, feelings, and habits. Neither vice nor crime, however extreme, is proof of Insanity. To be so, it must be proved through a chain of morbid symptoms to flow not from passion, but from disease; and attention should not, therefore, be entirely occupied by it, but should carefully traverse the whole affective life, in order to reveal the perversion of nature detectable in a case of real moral Insanity, and the connexion of the morbid change with an efficient cause of disease. A man may get into the police-court for stealing, in whom one may perceive instantly the earliest symptoms of general paralysis; another may commit murder, apparently without motive, or from a very inadequate motive, in whom a melancholic anguish has risen to a convulsive explosion; and a third may perpetrate violence in a state of affective derangement, which skilled observation recognises to be premonitory of, or vicarious of, an attack of epilepsy.

When it is a question of the existence of an irresistible impulse in any case of homicidal or other kind of violence, it is very necessary to keep in view the possibility of epilepsy, either in the form of epileptic vertigo or in its convulsive form. When an epileptic person has done a murder without discoverable motive, without advantage to himself or any one else, without premeditation, without malice, openly, it is almost certain that he has been driven to the crime by an irresistible impulse.

General paralysis is easily recognised after it has passed its earliest stage. It is not always easy of diagnosis before the physical signs appear; and yet a man may at this stage get into trouble—get into the police-court, or get married foolishly—entirely by reason of the disease. It is necessary to weigh carefully the character of the act, whether it is anywise explicable, or motiveless and quite unaccountable; to mark well the state of the patient's articulation under excitement or after a sleepless night; to note his happy and elated mood of mind; and to attend to the great exaggeration and general extravagance of his conversation on all matters concerning himself, even when there is no fixed and positive delusion. General paralytics, in the early stage, speak so extravagantly and absurdly regarding things which they have seen, or events in which they have been concerned, that an inexperienced person might be apt to put down the whole as a delusion. It is needful to bear in mind that there may be some foundation of fact in what they say of themselves—that they do not at first so much invent as outrageously exaggerate. It is needful also to remember the alternations of calmness and apparent sanity which occur in the early course of the disease. The main points to direct attention to are, first, any indications that there may be of altered speech and of loss of memory and mental power; and, secondly, the light in which the patient regards these symptoms when they are pointed out to him. If they are positively present, and there is on his part an entire unconsciousness of them, or if he laughs at and makes light of them, as is often the case, affirming that he was never better in his life, then it is almost certain that he is the victim of incipient general paralysis.

PATHOLOGY.—Though it may be that there are no morbid changes detectable in the brain of one who has died raving mad, it is none the less certain that, with change of energy, there is a correlative change in the nervous substratum. Nothing is yet known of the intimate constitution of nervous element, or of the mode of its functional action; and it is beyond question that the difference in its condition may be the difference between life and death, without any appreciable physical or chemical change. As the means of research improve, however, the instances of Insanity in which morbid changes are not found are more and more rare; and those who have most studied the matter are those who are most certain and speak most confidently of the invariable existence of such changes. When a morbid poison acts with its greatest intensity, there are notably fewer traces of structural alteration than where its action has been less intense and more prolonged; and, in like manner, appreciable organic changes in Insanity may be justly expected only when the degeneration has been extreme and long continued—in chronic mania, in chronic dementia, in general paralysis, and epileptic Insanity. Where this has been the case, morbid changes are seldom looked for in vain.

Investigations into the electrical properties of nerves, and into the phenomena of conduction by them, have not only rendered conceivable the existence of important, though undetectable, molecular changes among their ultimate elements, but have proved the necessity of dismissing all metaphysical conceptions of nervous function, and of making positive research into the physical and chemical conditions which, whatever its nature, determine its manifestations. So far from conduction by nerve being due to the instantaneous passage of some imponderable principle, it depends upon a modification of its molecular constitution, for which a certain time is essential; for it has been shown that a definite period of time, varying in different persons and at different periods in the same person, is necessary for the propagation of a stimulus from the peripheric ending of a nerve to its central ending in the brain; and when the stimulus has reached the brain, a certain time must elapse before the will can transmit a message to the muscles so as to produce motion. No investigation has yet been made of the time-rate of activity of the cerebral centres, but there is assuredly a considerable variation in the time in which the same mental functions are performed by different persons, or by the same person at different times. "The mind in health," says Locke, "will boggle and stand still, and one cannot get it a step forward, and at another time it will press forward, and there is no holding it in." Appreciable and variable, then, is the time-rate of thought, and the measure probably of that intimate molecular activity which is the condition of its manifestations. That such molecular activity does take place, the "waste" after function proves; the chemical reaction of nerve becomes acid after activity, owing probably to the formation of lactic acid in the retrograde metamorphosis; and the increase of phosphates in the urine, and the bodily exhaustion after great mental work, are only to be explained by supposing an idea to be accompanied by a correlative change in the nerve-cells. Here, indeed, is a region of most delicate activity, which, like that of thermal oscillations, or of undulations of light, is yet impenetrable to sense; and so far from its being improbable that undetected morbid changes may exist in Insanity, so far from its being wonderful that morbid appearances are sometimes not found, the wonder truly is that they should have been expected always. Where the subtlety of nature so much exceeds the subtlety of human investigation, to conclude from the non-appearance the non-existence of change, would be analogous to the blind man asserting that there are no colours, the deaf man that there are no sounds.

Not only have erroneous ideas been entertained respecting the kind of organic change that might suffice to give rise to Insanity, but the nervous element itself, as a living individual entity with intrinsic properties, has been commonly ignored; the main stress having been laid upon the blood-vessels, as if they were the primary agents in exciting and keeping up the mental disorder. The truth is, that the first step in Insanity often is, as it is in inflammation, a direct change in the individual elements of tissue, the change in the blood-vessels being secondary. The experiments of Lister[1] have proved that in the case of mechanical or chemical injury to some tissue, the individual elements are directly injured, and that a determination of blood, a dilatation of the vessels, and an adhesion of the corpuscles follow the local mischief; and it is easy to conceive that damage to the nervous element of the brain, however caused—whether from overwork or emotional anxiety, or from poison in the blood, or by direct injury or reflex irritation—may in like manner declare itself in disordered function, and be accompanied or followed by vascular disturbance. The nervous element is brought to a lower state of life, and manifests its deviation or degeneration from its normal kind by an abnormal or degenerate energy, while the disturbance of circulation takes place as a coincident or sequent effect of a common cause. Where there is hereditary taint, there is innate vice or defect in the constitution of nerve element, and it will accordingly break down more easily under adverse stress. The effects of strychnia may serve to illustrate the presumed course of events: when a dog is poisoned with strychnia, there may be no morbid appearances; but if there be any, they are such as congestion of the spinal cord, aneurismal dilatation of the capillaries, and perhaps small effusions of blood into the grey matter. The congestion is plainly a secondary result of the intense morbid activity of the directly poisoned nervous element. Here, then, is the abstract and brief chronicle of the order of events in many cases of Insanity. Transfer the convulsive action from the spinal cells to the cortical cells of the hemispheres, the result is a violent and acute mania in which the acute determination of blood is certainly not the cause, if it be not the consequence, of the degenerate function. In what is called *mania transitoria,* the patient falls suddenly into a violent fury, in which he raves and often exhibits destructive impulses; his face is flushed, his head hot, and there is plainly an active determination of blood to the brain; and in a short time the fury subsides, and the man is himself again, scarce conscious, or quite unconscious, of what has happened. Was the rush of blood the primary and active agent in the production of the fury? Was it not rather secondary to the intense morbid or degenerate action of the nervous centre; the attack representing a sort of epilepsy, and the congestion taking place not otherwise than as it takes place in the spinal cord poisoned by strychnia? So in chronic insanity, the congestion discoverable may not be the cause, but the effect and evidence of the morbid action of nervous element. And in those cases of Insanity in which there is no special morbid appearance after death, though there has been fixed delusion during life, it is because the definite morbid action which does exist takes place in that innermost region of activity of individual element to which our senses have not yet gained access. Only by fixing attention on the individual elements will a just conception be formed of the mode of that degeneration which reveals itself in mental disorder, but at present is not otherwise revealed; and only thus will the morbid appearances that are met with receive their right interpretation.

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[1] On the Early Stages of Inflammation, by Joseph Lister, F.R.S.; vol. xxxi. Philosophical Transactions, 1858.

It would be one-sided and mischievous, in another way, to overlook the fact that disturbances of circulation, of extraneous origin, may directly favour and even produce Insanity. Having regard to the extreme susceptibility of nervous element, and the abundance of the supply of blood to it, there can be no question that the quantity and quality of the blood play a weighty part in the pathology of Insanity.

Quantity and Quality of the Blood.—Since the time of Hippocrates, it has been known that similar symptoms are produced by too much and by too little blood in the brain. In that continued active relation between the blood and the nervous element, whereby due reparative material is brought and waste matter carried away, it amounts to much the same thing whether, through stasis of the blood, the refuse is not carried away, nor the supply brought to the spot where it is wanted, or whether the like result ensues by reason of a defective blood or a deficient circulation. Now, although temporary irregularities in the cerebral circulation may, and often do, pass away without leaving behind any abiding ill effects, yet when they recur frequently, and become more lasting, their disappearance is by no means the disappearance of the entire evil; they are efficient to initiate a degeneration, which thenceforth continues of itself and leads to permanent mental derangement. Once the *habit* of a definite morbid action is fixed in a part, it continues almost as naturally as, under better auspices, the normal physiological action.

A vitiated state of blood, by reason of matters bred in it, or introduced from without, may act perniciously on the supreme cerebral cells. The rapid recovery which takes place after moderate doses of alcohol, opium, Indian hemp, seems to show that the damage they inflict is transitory; but it admits of no question, that when nervous element is repeatedly exposed to their poisonous agency, it acquires a disposition to a degenerate function. The intense gloom produced by the presence of bile in the blood, and the extreme irritability produced by some urinary constituent in the blood of a gouty patient, serve to show what effects upon the supreme nervous centres may be due to the non-evacuation of the waste matters of the tissues. When, furthermore, it is remembered that the blood is itself a living, developing fluid; that, "burnished with a living splendour," it circulates rapidly through the body, supplying the material for the nutrition of the various tissues, receiving the waste matter of their activity, and carrying it to those parts where it may either be appropriated and so removed by nutrition, or eliminated by secretion,—it is plain that multitudinous changes are continually taking place in its constitution and composition, that its existence is a continued metastasis. There is the widest possibility, then, of abnormal changes in some of the manifold processes of its complex life and function, such as may generate products injurious or fatal to the life of nervous element. Poverty of blood undoubtedly plays a weighty part in Insanity, as in other nervous diseases; and there is, in the effects of the viruses of acute fevers, ample evidence that morbid poisons, bred in the organism, or entering it from without, may act in the most baneful manner on the nervous centres. In some cases of malignant typhus, and in the putrid infection after surgical operations, the virus generated is directly fatal to the life of nerve element; and when it acts with less intensity, it gives rise to the delirium of fever, and predisposes probably to the Insanity following acute fevers.

Reflex Action, or Sympathy.—The supreme cerebral centres may—like other nervous centres—suffer secondarily from morbid irritation in some other part of the body; though why they should do so at one time and not at another, we know not any more than why epilepsy should be caused by an eccentric irritation at one time and not at another. That they do so, many recorded instances clearly testify.[1] When a chronic insanity is brought about in this way, the delusion has sometimes a relation to the primary morbid cause; the secondary derangement of the cerebral centres testifying to the special effect of the particular diseased organ—as, for example, when a woman, with uterine or ovarian disease, believes she is with child by supernatural means, or, with morbid irritation of the sexual organs, has salacious delusions. There is the most perfect harmony, the most intimate connexion or sympathy, between the different organs of the body, as the expression of its organic life—a unity of the organism beneath consciousness; and the brain is quite aware that the body has a liver or a stomach, and feels the effects of disorder in any one of the organs, without declaring it directly in consciousness. This unconscious, but not unimportant, cerebral activity, which is the expression of the organic sympathies of the brain, receives its most striking illustrations in the influence on the mind of the development of the sexual organs at puberty, and in their subsequent influence on dreams; and it is of great weight in the production of morbid mental phenomena. A just appreciation of its importance will not fail to teach the lesson which a true conception of the organism as an individual whole, formed of differentiated parts harmoniously co-ordinated, teaches also, that every organic motion, visible or invisible, sensible or insensible, ministrant to the highest aim or to the humblest function, does not pass issueless, but has its due effect upon the whole, and is felt throughout the most complex recesses of the mental life.[2]

The primary morbid centre, which gives rise to secondary derangement by a reflex or sympathetic action, may not be in a distant organ; it may be in the brain itself. A tumour, abscess, or local softening does not interfere with the mental processes at one time, while it produces the gravest disorder of them at another; and it is not uncommon in abscess of the brain for the mental symptoms, when there are any, entirely to disappear for a time, and then to return suddenly in all their gravity, the derangement or abolition having been due to a sympathetic or reflex action.

Here, then, should be distinguished the different kinds of disorder of the cerebral centres to which a morbid cause may give rise. The sudden appearance and equally sudden disappearance of extreme mental derangement prove that it is functional; it being impossible to conceive the existence of serious organic change in such case. Although, then, the functional disorder necessarily implies a molecular change of some kind in the nervous element, the change may well be one affecting the polar molecules, such as the researches of Du Bois Reymond have proved may rapidly appear and rapidly disappear. At any rate the induction, by definite experiments, of recognisable transitory changes, in the physical constitution and the function of nerve, warrants the belief in similar modifications producible by morbid causes which are not artificial, but which are just as abnormal as if they were artificial. This modification of molecular constitution, which vanishes at first with the removal of the cause, will not fail, if too great or too prolonged, to degenerate into actual nutritive change and structural disease, just as an emotion which alters a secretion temporarily may, when long enduring, lead to nutritive change in the secreting organ.

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[1] References to such cases may be found in Griesinger's work. There is a remarkable case also related by Dr. Brown-Séquard in his Lectures on the Physiology and Pathology of the Nervous System. 1860.

[2] "Man is all symmetrie,
Full of proportion one limb to another,
And all to all the world besides.
Each part calls the furthest brother;
For head with foot hath private amity,
And both with moon and tides."—George Herbert.

Excessive functional activity.—The display of function being the waste of matter, it is plain that if there be not due intervals of periodical rest, degeneration of nervous element must take place as surely as when directly injured by morbid poison, or mechanical irritant, or as surely as a fuelless fire must go out. It is sleep which thus knits up the ravelled structure of nervous element; for during sleep, organic assimilation restores as statical or "latent" the power which has been expended in function, or made "actual" in energy. Sleeplessness is, accordingly, one of the troubles following intense mental anxiety, or too great mental activity, and forerunning Insanity: that which should heal the breach is rendered impossible by the extent of the breach. Like Hamlet, according to Polonius, the individual falls into a sadness, thence into a watch, then into a lightness, and by this declension into the madness wherein he finally raves. To provoke repose in him is the prime condition of restoration; the power of its "closing the eye of anguish," and healing "the great breach in the abused nature" of nervous element.

Thus much from a pathological point of view concerning the causation of Insanity: it now remains to enumerate the morbid appearances that have been met with in the brain and its membranes.

MORBID ANATOMY.—The broad result of investigation is, that the morbid changes most constantly met with are such as affect the surface of the brain and the membranes immediately covering it; and of these changes, those in the layers of the cortical substance are the principal and essential. The signs of more or less inflammation of the membranes, especially milky opacities of the arachnoid, are common enough in the bodies of those who have not died insane. But there would appear to be some hindrance to inflammation spreading easily to adjacent parts that are of different structure; whereas, when they are of the same structure, it passes readily from element to element of the same kind, as it were by an infection; the intercostal muscles are scarcely affected in acute pleurisy, the muscular walls of the intestines scarcely affected in peritonitis, and inflammation takes place in the membranes of the brain, without seriously implicating the cortical layers. If these are involved, there can be no question that the mind suffers. Deaths in the acute stage of Insanity are not usual; but if an opportunity presents itself of examining the brain at this early stage, the morbid appearances are those of acute hyperæmia—namely, great injection of the pia mater, with spots of ecchymosis, and more or less discoloration and softening of the cortical layers, which may be separated easily with the handle of a scalpel from the white substance beneath; the discoloration being in red streaks or stains, with spots of extravasated blood, and the softening of a violet or pinkish hue; the puncta vaculosa of the white substance are also increased. There are no observable differences between the morbid appearances met with in acute mania and in acute melancholia; and though such fact ill agrees with their different symptoms, it is not entirely singular, forasmuch as alcohol makes one man lively and another melancholic. A differential pathology would involve the knowledge of what constitutes individual disposition or temperament. It must be confessed that in both mania and melancholia morbid changes are sometimes wanting.

The instances of chronic insanity in which no morbid lesions appear, are rare: the longer the disease has lasted, the more evident they usually are. There is mostly some degree of thickening or opacity of the arachnoid, which may form a white opaque layer through which the convolutions are scarce visible; and many of the more advanced cases exhibit some degree of atrophy of the brain, especially of the convolutions—these appearing shrunk, pale, and anæmic, or as if some were wanting and replaced by an effusion of clear serum into the subarachnoid space. The atrophic change may be, according to Dr. Wilks, a simple degeneration, or a degeneration associated with the changes resulting from chronic inflammation. The pia mater is at times adherent to the surface of the brain, so that there is some difficulty in stripping it off without bringing portions of grey matter away with it. This adhesion is not peculiar to general paralysis, as some have thought, though most often met with in it; for it is now and then found in other forms of chronic insanity, particularly those following epilepsy and drunkenness. The ependyma of the ventricles is thickened, and sometimes covered with fine granulations, such as have been described also by L. Meyer on the arachnoid and inner surface of the dura mater.[1] Dr. Wilks has seen a minutely granular condition of the lining of the ventricles, in a case of acute mania; he has often seen it in epilepsy—once, when the granules were as large as peas, and "the whole surface of the ventricles had very much the appearance of an ice-plant." In some cases, the exudation is in flattened scales or patches.[2]

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[1] Virchow's Archiv, B. xvii. s. 209.

[2] Clinical Notes on Atrophy of the Brain, by S. Wilks, M.D.; Journ. Ment. Science, October 1864.

The morbid changes most frequent in general paralysis, though in rare instances there are scarcely any detectable, are great œdema of the membranes, adhesion of the pia mater to the grey substance beneath, local discoloration or softening of the cortical layers, or superficial induration of them. More or less atrophy of the whole brain, and particularly of the convolutions, is common, and is accompanied with greater firmness of its substance, enlargement of the ventricles, and serous effusion into them. Diffuse pachymeningitis, effusion of blood into the membranes, or rather into the layers of exudation (Virchow, Rokitansky), and degeneration, atheromatous and calcareous, of the arteries, are not unfrequently met with. Though these changes are more common in general paralysis than in any other form of Insanity, they are by no means peculiar to it, nor are they constant in it; in some cases, the evidence of meningitis is most marked, in others that of atrophy.[1]

A diffuse albumino-fibroid exudation of low form, glueing the membranes to the surface of the brain, has been declared by some to be characteristic of syphilitic insanity. Instead of being diffused, the *gum-like* exudation, or *syphiloma,* as it has been called, may be circumscribed so as to form a tumour, and press into the substance of the brain, causing softening immediately around it; or again, it may be met with as a diffuse infiltration or a tumour within the brain, the membranes being unaffected. At the outset it consists of an exuberant growth of connective tissue, which afterwards undergoes more or less fatty degeneration; and it certainly has not any character by which it can be distinguished as a specific product.[2]

Researches have been made into the absolute weight and specific gravity of the brain in Insanity, but they have not been sufficiently exact.[3] Dr. Skae and Dr. Boyd found the absolute weight to be slightly increased in the insane, the increase being greatest in mania, and least in general paralysis. The specific gravity is also increased, the lowest specific gravity, which is still above the average, occurring in dementia, the highest in epilepsy. Dr. Bucknill observed that the mode of death influenced the results, and found also that the increase of specific gravity was due, in some cases, to a deposit of an inert albuminous matter amongst the proper nervous elements, and the consequent shrinking of these,—a condition seemingly not unlike that since described by Prof. Albers as *parenchymatous infarction of the brain,* and met with after typhus, in some cases of old Insanity, and in imbecile children, especially those of a scrofulous habit of body. A fibrinous or albumino-fibroid exudation would appear to be a not uncommon result of the degeneration of extreme Insanity; it is the condition probably of the increased consistency, or *sclerosis,* which is the final result of atrophy; and it is comparable with the product of what is described as chronic inflammation in other organs, as the liver and spleen. A similar exudation is the cause of the so-called hypertrophy of the brain from which children sometimes die, and which is now and then met with in single cases of Insanity and epilepsy.

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[1] A compact account of the morbid changes in general paralysis, with exhaustive references, will be found in a paper by Dr. E. Salomon on the Pathological Elements of General Paresis; Journ. Ment. Science, October 1862.

[2] Des Affections Nerveuses Syphilitiques, par Dr. Léon Gros et E. Lancereaux, 1861. Ueber die Natur der Constitutionell-syphilitischen Affectionen, von Rudolf Virchow; Archiv, B. xv. Das Syphilom, oder die Constitutionell-syphilitische Neubildung, von E. Wagner; Archiv der Heilkunde, 1863. Ueber Constitutionelle Syphilis des Gehirns, von Dr. Ludwig Meyer; Zeitschrift f. Psychiatrie, 1861. Des Affections Nerveuses Syphilitiques; M. Zambaco. Wiener Medizinal-Halle Zeitschrift, February 1864; Dr. Leidesdorf. Zeitschrift f. Psychiatrie, 1863; Dr. Westphal.

[3] Dr. Bastian's recent researches seem to prove that the specific gravity of different parts of the grey matter differs considerably in health. "On the Specific Gravity of the Human Brain;" Journ. Ment. Science, January 1866.

The microscope has of late years added something to our knowledge of the nature of the morbid changes in Insanity. The most constant result has been to establish a rank or exuberant growth of connective tissue, and a coincident or sequent decay or destruction of the proper nervous elements, in Insanity of long standing, and especially in general paralysis. The researches of Rokitansky and Wedl reveal a more or less diseased state of the capillaries of the cortical substance in general paralysis: these exhibit aneurismal dilatations, and tortuosities varying from a single twist to a more complex twisting and even to little knots of varicose vessels.[1] Round the capillaries, small arteries, and veins there is a hyaline deposit of embryonic connective tissue beset with oblong nuclei; this afterwards becoming more and more fibrous, so that the vessel may look like a piece of connective tissue, in which granules of fat or calcareous matter ultimately appear. Other products of the retrograde metamorphosis, such as amyloid corpuscles and colloid corpuscles, are also found in connexion with the hypertrophied tissue, which, whether called inflammatory or not, is itself essentially the result of a vital degeneration. The degeneration appears to be of two kinds: first, there is a defective nutrition, a retrograde nutritive process, whereby the vitality not reaching the height necessary for the production of the proper elements of the structure, there are engendered from the germinal nuclei elements of a lower kind—connective tissue instead of nerve; and, secondly, there is a retrograde metamorphosis of the formed elements of the part—a colloid, fatty, or calcareous degeneration. Be this as it may, there are at any rate three principal stages of the degenerative process: (1), a morbid change in the vessels, whereby there must be a great hindrance to regular nutrition; (2), atrophy of nerve element, either in consequence of the hindrance to nutrition (Rindfleisch[2]), or from the rank growth of connective tissue (Rokitansky); (3), the increase and subsequent retrograde metamorphosis of connective tissue. Recently it has been asserted by Dr. Tigges, that there is, even at an early stage, an increase of nuclei in the ganglionic cells; the numerous scattered nuclei, usually thought to belong to connective tissue, he considers to have escaped from ganglionic cells at a later stage of their inflammatory degeneration.[1] Such changes are not peculiar, as some have said, to general paralysis; like changes have been described by Wedl[2] in the brains of three congenital idiots, and have been met with in dementia following on long-continued Insanity, and in tabes dorsalis; and there can be little doubt that the morbid product in syphilitic dementia is of a similar nature.

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[1] Ueber Bindegewebeswucherung im Nervensysteme; Rokitansky, 1857. Wedl, Beiträge zur Pathologie der Blutgefässe; Wien, 1859.

[2] Histologisches Detail zu den grauen Degeneration von Gehirn und Rückenmark. Von Dr. E. Rindfleisch. Virchow's Archiv, B. vi.

Briefly summed up, then, the kinds of degeneration met with in the brain, after Insanity, are as follow:—1. There is in acute insanity an acute hyperæmia, or the early stage of *inflammatory degeneration*. 2. There is that degeneration which consists in the increase of connective tissue, and in the atrophy of the nervous elements, and which might be called *connective tissue degeneration*. Whether called sub-inflammatory or not, is not of much moment, so long as we keep in mind the true relations of organic element to the supply of blood, and the degenerate nature of inflammation, whether acute or chronic. 3. *Fatty degeneration* takes place not only in the small vessels, as in atheroma, and in the new morbid products, but also in the broken-up nervous elements, and even in the nerve-cells. 4. The *amyloid degeneration* is undoubtedly pathological. Wedl holds that the amyloid corpuscles should be ranked along with the so-called colloid bodies, and viewed as the result of an increased exudation that may take place without hyperæmia. Rindfleisch, on the other hand, believes that he has traced their production from the nucleated connective-tissue corpuscles; while some, like Rokitansky, maintain that the ganglionic cells themselves are converted into colloid bodies. 5. *Pigmentary degeneration* is sometimes met with. In senile atrophy the ganglionic cells are sometimes richly filled with brown pigment molecules; and, in one case of dementia, where there was partial paralysis of the tongue, Schroeder van der Kolk found the cells forming the nuclei of the hypoglossal nerves in such a state of blackish-brown degeneration that he at first mistook them for little points of blood, but they were filled with granular, dark-brown pigment. Dr. Lockhart Clarke has observed similar pigmentary degeneration of the cells in general paralysis. It is worthy of remark, that cases of pigmentary degeneration of the retina are often found to occur in the same family, to be accompanied with general defective development, mental and bodily, occasionally with mutism and Cretinism, and to occur in those who, like albinos, are sometimes the degenerate offspring of marriages of consanguinity.[3] 6. *Calcareous degeneration* is common enough in the hypertrophied connective tissue and in the small blood-vessels; and it has been observed in the ganglionic cells themselves. Erlenmeyer met with calcified cells in the optic commissure of a monomaniac; Förster figures calcified cells found in the grey substance of the lumbar enlargement of a boy whose lower extremities were paralysed; Heschl found what he calls ossified cells in the brain of a patient who died melancholic; and Wilks believes certain bodies, found in the brain of a general paralytic, in whom the small arteries were calcified, to have been ganglionic cells that had undergone calcareous degeneration.

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[1] Zeitschrift für Pscyhiatrie, B. xx. In Virchow's Archiv, 1865, Dr. Franz Meschede has striven to prove that hyperæmia and parenchymatous swelling of the inner layer of the cortical substance are the beginning, and fatty degeneration the end, of the organic changes in general paralysis.

[2] Histologische Untersuchungen über Hirntheile dreier Salzburger Idioten. Von Prof. C. Wedl; Medizinische Jahrbücher der K. K. Gesellschaft der Aerzte in Wien. 1863. Heft 2 und 3.

[3] On Retinitis Pigmentosa, by J. Laurence; Ophthalmic Review, No. 5, April 1865. The observations of Prof. Graefe and Liebreich are quoted.

Those who duly weigh the pathological import of the kinds of degeneration enumerated, who reflect on the great gap which there is between a calcareous granule and a nerve-cell in the economy of nature, or between a connective-tissue corpuscle and a nerve-cell in the histological scale of life, must admit that the difference is not less great than that between dementia and sound mental action, and will scarce venture to assert that the morbid appearances throw no light whatever upon the nature of Insanity. Even the comparatively slight signs of hyperæmia are of weighty significance, if their true relations are recognised, if they are viewed as results and evidence of that degeneration of individual nervous element, of which the mental disorder is also result and evidence, if they and the Insanity are viewed as, what they often are, concomitant effects of a common cause.

PROGNOSIS.—Two questions at once present themselves: the first, whether the disease directly endangers life; the second, and perhaps more solemn one, whether there is any prospect of recovery. Respecting the first question, it may be said in general terms that Insanity does certainly reduce the mean duration of life, and much more so in its recent acute forms than in its more chronic forms. Of all forms, general paralysis is the most fatal, other varieties not being, as a rule, directly dangerous to life. Still, certain cases of acute mania and acute melancholia do terminate suddenly in death, owing probably to exhaustion, and it may be utterly impossible to say beforehand whether they are going to do so or not. When the temperature of the body rises several degrees above the natural standard, the prognosis is bad; and any indication of motor paralysis, or any kind of hybrid epileptiform convulsion, in the course of the disease, is of evil omen, while an attack of genuine epilepsy, unfavourable as regards recovery, is not so as regards life. A long-enduring refusal of food may sometimes end fatally, both in mania and melancholia.

What prospect there is of recovery in a particular case will depend greatly upon the cause of the attack, upon its form, and upon its duration. The more recent the outbreak, the better the chance of recovery, the expectation of which from proper treatment adopted within three months from the commencement is about four to one, while it is less than one to four after twelve months' duration of the disease. Certainly there do occur instances in which patients recover after being insane for years, but they are exceptional. When the stage of secondary dementia, incoherent or apathetic, has been reached, all hope of recovery is gone. Primary dementia is generally curable.

Of the acute primary forms of mental disease, melancholia is more curable than mania, although some have thought otherwise, deceived probably by the experience of an asylum into which simple cases of melancholy do not usually come. Next to melancholia acute mania is most curable; but when attacks of melancholia and mania alternate, the prognosis is very unfavourable. A day of great depression and weeping intervening in the course of acute mania, is of good omen. When the maniacal fury is subsiding, the prospect is good if the patient is sad and depressed, begins to inquire about his family, and to show other signs of a return to his former feelings; it is bad if the feelings remain unmoved, and the intellect is calm in its disorder—if, in other words, there is evidence of the organization of disorder. Even the disappearance of intellectual disorder is not a certain sign of recovery, unless there is a return to the old healthy feelings, and the patient recognises that he has been mad. A periodical recurrence of attacks of Insanity, with long intermissions, is of decidedly unfavourable augury; the attacks commonly become longer, the intermissions briefer, and the outlook gets more and more gloomy.

Monomania is far less curable than mania, the fixed delusions marking the establishment of a definite type of morbid action of a chronic nature. Recovery does sometimes take place under the influence of systematic moral discipline, or from a great shock to the system, whether emotional or produced by some intercurrent disease. In melancholia, where there is a fixed delusion that the cause of misery is in some external agency, the prognosis is unfavourable; but it is more favourable in the melancholic who attributes his affliction to his own imaginary backslidings. In like manner the homicidal melancholic, who believes himself the victim of persecution, seldom recovers; the suicidal melancholic, who is not so in consequence of any definite delusion, frequently does recover, especially after some serious and almost successful suicidal attempt.

In moral insanity the prognosis is bad; for these symptoms usually mark the tyranny of a bad organization. General paralysis may be pronounced incurable.

When Insanity has been slowly developed, the prognosis is more unfavourable than when it has been of sudden origin—this probably being a part of the larger truth, that when Insanity is slowly developed it is produced by the egoistic passions as an exaggeration of some peculiarity of character, as pride, ambition, avarice; but when it is suddenly caused, it is by the shock of an altruistic emotion, as, for example, grief about others. For a like reason a frequent alternation of active symptoms is more hopeful than a steady persistence in a particular group of quiet symptoms. Hereditary insanity is generally deemed most unfavourable, but recent researches prove that it is not so much so during a first attack, although the disease is more liable to recur than when not of hereditary origin. In the acute mania sometimes produced by drunkenness we may reasonably look for recovery, but by no means so in those cases in which a continued intemperance has resulted in mental weakness, loss of memory, and loss of energy of character. When Insanity has been caused by habits of self-abuse or by sexual excesses, the prognosis is unfavourable in all but the earliest stages. If religious excitement purely has been the cause of an outbreak, the prognosis is most favourable; but it is necessary to bear in mind that a form of religious insanity is the vicarious satisfaction of unsatisfied sexual love, that more or less nymphomania is oftentimes associated with it, and that the outlook then is hopelessly bad. When disease of brain, or injury of the head, or epilepsy, has been the cause, the derangement is practically incurable; but where it occurs during the decline of some acute disease, it is generally soon curable. The prognosis is bad in Insanity after sunstroke; nevertheless Dr. Skae mentions one case under his care in which recovery took place, being one of the very few recoveries on record from Insanity produced by this cause. The prognosis is favourable in hysterical insanity; as it is also in puerperal mania, in the insanity of pregnancy, of lactation, and of the climacteric change. A decidedly bad symptom is a fixed hallucination, as is also a complete preservation of bodily health with persistence of mental disorder; when there is palpable bodily disorder, as digestive disturbance, anæmia, menstrual irregularity, there is good hope that with the restoration of bodily health the mind may be restored also.

When Insanity has followed the suppression of an eruption or an accustomed discharge, the prognosis is favourable; when associated with phthisis, it is unfavourable as regards recovery and as regards life.

The most favourable age for recovery is youth, the probability of it diminishing with the advance of age, and few recovering after fifty: as many as 86 per cent. of males and 91 per cent. of females, attacked with mania under twenty years of age, recovered at the Somerset Asylum. This proportion will appear the more favourable when we reflect that children under ten or twelve years of age do not recover from mental derangement, it being often dependent upon defective organization, and associated with epilepsy. The recoveries amongst women exceed those amongst men, by reason probably of the frequency and fatality of general paralysis among men.

The general conclusion of Dr. Thurnam from his careful statistics is that, "as regards the recoveries established during any considerable period—say twenty years—a proportion of much less than 40 per cent. of the admissions is under ordinary circumstances to be regarded as a low proportion, and one much exceeding 45 per cent. as a high proportion." The liability to recurrence of the Insanity after recovery from the first attack cannot, he thinks, be estimated at less than 50 per cent., or as one in every two cases discharged recovered. On the whole, then, he holds that, of ten persons attacked, five recover and five die sooner or later. Of the five recoveries, not more than two remain well during the rest of their lives; the others have subsequent attacks, it may be after long intervals, during which at least two of them die.[1]

THERAPEUTICS.—The treatment of Insanity is moral and medical, the two methods properly being combined. It must again be *individual,* as the case is; for the varieties of the insane character demand different moral means, as the varieties of causes call for different medical treatment. It is necessary to penetrate the individual character, with the design of influencing it beneficially, and carefully to investigate the concurrence of conditions that have issued in Insanity, with the object of removing them. Herein lies the chief difficulty of treatment; in no other disease are there so much concealment and so much misrepresentation, witting and unwitting, on the part of friends. It is before all things necessary again that treatment should be begun early, before the habit of a definite morbid action has been fixed; but, though early, it must not be rashly vigorous and energetic, with the aim of effecting any sudden revolution, but rather patient and systematic, in the hope of a gradual change for the better. Whilst in other diseases time is reckoned by hours and days, it must in Insanity be reckoned by weeks and months.

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[1] In an elaborate paper on Vital Statistics and Observations, in the Journal of Mental Science, January 1865, Dr. Boyd records the statistics of the Somerset Asylum for many years:—

	Of 1,000 Males.	Of 1,000 Females.
Recovered	252	276
Relieved	55	79
Not improved	47	35
Died	324	258
Remaining	192	223
	870	871
Re-admissions	130	129
	1000	1000

The causes of death in 519 cases out of 2,000—295 males and 224 females—in which post-mortem examinations were made, were as follow:—

		Males.	Females.
Diseases of	Respiratory organs in	148	104
,,	Nervous system	112	73
,,	Digestive organs	18	41
,,	Vascular system	11	18
,,	Genito-urinary	2	1
,,	Locomotor organs	1	3
,,	Fevers	0	2
,,	Accidents	3	2
		295	244

The diseases of the respiratory organs which proved fatal, were principally pneumonia and phthisis.

Moral Treatment.—To remove the patient from the midst of those circumstances under which Insanity has been produced, must be the first aim of treatment. There is extreme difficulty in treating satisfactorily an insane person in his own house, amongst his own kindred, where he has been accustomed to exercise authority, or to exact attention, and where he continually finds new occasions for outbreaks of anger or fresh food for his delusions. An entire change in the surroundings will sometimes of itself lead to recovery; if the patient is melancholic, he no longer receives the impressions of those whom, having most loved when well, he now most mistrusts, or concerning whom he grieves that his affections are so much changed; if he is maniacal, he is not specially irritated by the opposition of those whose acquiescence he has been accustomed to, or encouraged by their submission to his whims and their indulgence of his follies. Travelling may be recommended in the early stages, in order to secure change of place and scene; or if the patient cannot travel, he may be removed from his own home to another residence, and systematically treated there. If the pecuniary means do not admit of the adoption of either of these expedients, or if the patient is furious, or desperately suicidal, or persistently refuses food, it will be necessary to send him to a suitable asylum. It must be borne in mind that an insane person cannot, from the very nature of his disease, have his own way, and that to allow him to do so would be directly to aggravate his disease. To put him under restraint in some way, to exercise for him that control which he cannot exercise for himself, is indispensable so long as there is a hope of cure: to let him distinctly understand that this is legally done will of itself have a beneficial effect. The melancholic who finds himself in an asylum finds a real grief to alternate with or perhaps to take the place of his fancied affliction; and the maniacal patient, feeling his wild spirit of exultation to be rudely checked by the influence of a systematic control, can scarce fail to have more sober reflections aroused. It is extremely objectionable except in an emergency to entrap a patient into an asylum; there should be no deception about the matter, if possible, but all should be done openly and firmly, in the spirit in which an act of obedience is inculcated upon a child, and in any case inflexibly insisted upon.

The patient having been removed from those influences which have contributed to the production of the disease and tend to keep it up, and having been made to recognise from without a control which he cannot exercise from within, it remains to strive persistently and patiently by every inducement to arouse him from his self-brooding or self-exaltation, and to engage his attention in matters external—to make him step out of himself. This is best done by engaging him earnestly in some occupation or in a variety of amusements; and this will be more easily done now that the surroundings are so entirely changed. The activity of the morbid thoughts and feelings subsiding in the new relations and under new impressions, more healthy feelings may be gradually awakened; and the activity of healthy thought and feeling will not fail in its turn further to favour the decay of morbid feeling. It is not by arguing against a delusion or directly contradicting it that any good will be done: it were almost as well to argue against the east wind or a convulsion; but by engaging the mind in other things, and substituting a healthy energy for the morbid energy, this will be most likely to abate and finally die out. But though it is of little use talking against a delusion, it is desirable to avoid agreeing with it: by quiet dissent or a mild expression of incredulity when it is mentioned, the patient should be made to understand clearly that he is in a minority of one. It is most necessary to avoid any harsh word or act, a tone of ridicule, or a look of disgust; an angry speech will often be remembered when the frenzy has passed off, and will leave a sore feeling in the patient's mind. It is a mischievous and silly practice too to speak to insane persons as if they were babies: they are often more sensitive to such impertinences than sane persons, while they seldom fail to be influenced beneficially by a sincere, sensible, and sympathetic address.

Medical Treatment.—There is no specific agent in the treatment of any form of Insanity. A truly scientific treatment will be grounded on the removal of those bodily conditions which may appear to have acted as causes of the disease and on the general improvement of nutrition. An attack of melancholia occurring in a gouty subject, and perhaps taking the place of an attack of gout, is sometimes cured by the treatment proper for gout; and there are cases on record in which acute mental derangement has affected persons living in a malarious district in regular tertian or quartan attacks, and has been cured by quinine. The morbid sensations, so common in Insanity, should not be overlooked, as they often arise from some real bodily derangement, and tend to keep up the delusion. Now, bodily disease is not always easily detected in the insane; for the usual symptoms are very much masked, and they, like animals, make no intelligent complaint. "Insanity," says Dr. Bucknill, "is not confined to the brain, and when it is confirmed, a man becomes a lunatic to his finger ends." It is necessary, therefore, to examine carefully into the state of the different bodily functions and to pay particular attention to the physical signs of disease: there may be no cough, no expectoration, when the thermometer or the stethoscope reveals advancing phthisis.

General bloodletting is now rarely, if ever, used; even in the most acute and seemingly sthenic insanity it is not simply useless, but it is positively pernicious. Violent symptoms may abate for a time, but the disease is very apt to become chronic and to pass rapidly into dementia. Local abstraction of blood by leeches or by cupping may be useful where there appears to be great determination of blood to the brain; by withdrawing blood from the overloaded vessels the opportunity of rest is afforded to the struggling and suffering nervous element. It may be well to add here that in Insanity it is not judicious but mischievous to shave the patient's head, to confine him to bed, to shut out the light, and to treat him in all ways as if he were suffering from an acute inflammation of the brain or its membranes; on the contrary, in almost all cases it is necessary to enjoin abundant exercise in the open air.

The continued application of cold to the head by means of a douche pipe, or by pouring cold water upon it, while the patient lies in a warm bath, is often successful in calming excitement and in procuring sleep in acute insanity. The warm bath alone, taken for about half an hour, has a soothing effect, and may induce sleep; and its efficacy has been supposed to be wonderfully increased by the addition of several handfuls of mustard, so as to produce a general redness of the surface of the body. Brierre de Boismont professes to get very good results from employing the warm bath for eight or ten hours at a time; and Leidesdorf has used for three or four hours, and in many cases with marked calming effect, a bath constructed by Professor Hebra, in which patients may be kept night and day at a definite temperature. Such a use of the bath must obviously be avoided where the pulse is feeble and where there is anything like commencing paralysis, and it is of no avail in cases of chronic insanity. The prolonged use of the shower bath and of the cold bath, at one time much in fashion, is now justly abandoned. The shower bath or cold douche may certainly be usefully employed in certain cases of melancholia, where reaction does not fail to take place after it, and in cases of chronic insanity with the purpose of rousing the patient and giving tone to the system; but its use should never be continued for more than three minutes, and it should not be with the aim of producing any special effect, but on general principles of improving the bodily health. Pouring water on the head from a hand shower bath, or a common garden watering-pot, or sponging the forehead and face with cold water, is refreshing and grateful to the patient suffering from acute excitement, and has sometimes a good effect beyond what might be expected from a measure so simple. The virtues of the Turkish bath were at one time much vaunted by its advocates, but an exact discrimination of the cases in which it is useful yet remains to be made. Packing in a wet sheet has sometimes a beneficial effect, and is commonly rather grateful to the patient.

Counter-irritants are not much used now in Insanity. Schroeder van der Kolk, however, put much faith in the application of strong tartar-emetic ointment, or of a blister to the shaven scalp; and Dr. Bucknill has found it useful to rub croton oil into the scalp in the passage from acute to chronic insanity or dementia, and in chronic melancholy with delusion. Blisters to the nape of the neck appear to have little other effect than to increase mental irritation; and the benefit of setons and issues in the neck is very problematical.

After errors of digestion and secretion have been duly attended to, the diet of the insane should be good; and it will be desirable in most chronic cases, and in many acute cases, to allow a moderate use of wine. There can be little doubt that an attack of Insanity might sometimes be warded off by a generous diet and free use of wine at a sufficiently early stage. It is at any rate a truth worthy of all acceptation, that energetic antiphlogistic treatment in the course of Insanity is energetic mischief. Leeches may be applied to the head, and a patient may be kept on low diet, in order to subdue maniacal excitement, without any other result than an increase of the excitement with the increase of exhaustion; and the most active purges may be given, and given in vain, to overcome an obstinate constipation,—when brandy and beef-tea, reducing exhaustion, will subdue excitement, and a simple enema will produce full action of the bowels. Active purgation, once so much favoured, is now quite eschewed in all forms of Insanity. The bowels may generally be regulated by dietetic means; and if a purge is needed, a dose of aloes, rhubarb, or castor oil will answer every purpose—a moderate dose of the last often succeeding where the most drastic purgatives fail. The state of the bowels ought, however, to be carefully attended to, for melancholia has sometimes been produced by habitual constipation, and is always made worse by it. A mixture containing sulphate of magnesia, sulphate of iron, with a little quinine and sulphuric acid, will be found very useful. It will, of course, be necessary to be guided by the bodily state of the patient, and by the history of the causation of the disease, as to whether wine is given or not in the most acute stage; it may be desirable in cases of a sthenic type to do nothing but wait patiently, only preventing the patient from doing harm to himself or others, until the fury of the storm has passed, and then to give support.

Amongst drugs useful in Insanity, opium undoubtedly occupies the first place. It is especially useful in that state of mental hyperæsthesia which so often precedes the outbreak of Insanity: when the mental tone is so changed that almost every impression is painful, then opium, freely administered, has virtues which can scarce be exaggerated. It is useful, again, in cases of simple melancholia, when it should be given in doses of one, or even two, grains twice a day, and continued steadily for weeks, notwithstanding an apparent want of success at first. In these cases it does not usually produce constipation; but if it does, then each dose may be combined with a grain of extract of aloes or a quarter of a grain of podophyllin. Where there is fixed delusion of some standing, it is not of much use, except as an occasional expedient for procuring sleep.

As opium agrees better with persons of melancholic than with those of sanguine temperament, so it appears to be more useful in melancholic than in maniacal forms of Insanity. The early writers on Insanity condemned the use of opium in mania, because of the exaggeration of the mental symptoms which they observed in some cases after sleep occasioned by it. Certainly there are cases of acute mania in which, after two or three hours of such sleep, the patient seems only to have gained new vigour for a fresh start in frenzy, and in which it is impossible to help feeling that the drug has done harm rather than good. But there are, on the other hand, cases in which there can be no reasonable doubt of the benefit conferred by opium. These are the cases in which there is no heat or congestion of the head, but where the face is pale, the pulse weak, and where a restless activity and incoherence are accompanied with want of sleep. In the mania caused by intemperance, in the mania or delirium of nervous exhaustion, and in puerperal mania, opium is beneficial; but it is of little use in melancholia with stupor, in idiopathic sthenic mania, or in the attacks of excitement that occur in the course of chronic mania and general paralysis.

In cases of great excitement, maniacal or melancholic, where opium does no good, large doses of digitalis sometimes produce the best effects, especially where there is general excitement of the circulation. The excitement abates, and the pulse, falling in frequency, may be kept for a long time at a standard below the average. In the attacks of excitement which occur in the course of general paralysis, the effects of digitalis are excellent; a single dose of two drachms, or continued doses of half a drachm every three or four hours, being safely given. Hydrocyanic acid may be usefully combined with it; and some entertain a high opinion of the good effects of this acid when given by itself.

The sub-cutaneous injection of morphia is a valuable expedient to have recourse to, where there is a refusal to take medicine, and it usually operates much more effectually than opium taken by the mouth. Not more than a quarter of a grain should be injected to commence with, and the quantity may subsequently be increased, if necessary. It will be well to have in mind, that neither opium by the mouth, nor morphia hypodermically injected, will quench the fury of acute mania, and that successive doses of opium or successive injections of morphia, followed by brief snatches of fitful sleep, have been followed also by fatal collapse.

It is not yet possible to speak positively and definitely of the virtues of chloral as a remedy in Insanity. So far as my experience reaches, it is that, given in doses of 30 grains, repeated in an hour or two if necessary, it will produce sleep in most cases of Insanity, but that permanent good follows the sleep only in those asthenic cases in which opium does good. There can be no question that we have in this drug a useful hypnotic, but it is another question whether hypnotics will stay the course of acute Insanity.

Hyoscyamus is useful also in some cases where opium does not agree, but it should be given in doses of not less than a drachm to begin with. Tartar emetic will often calm for a time the most furious maniac, through the prostration which it produces, but it does no permanent good, and its employment for such purpose is rather a relic of the old system of quieting a patient by some violent means or other, short of actually killing him. If mercury be ever useful, and not mischievous, in the treatment of Insanity, it is when given in small doses of the perchloride, in cases that are becoming chronic, or where there is a suspicion of syphilis. To administer mercury systematically in general paralysis, as has been done, is as unaccountable in theory as it is undoubtedly pernicious in practice.

In hysterical mania, in epileptic mania, and in mania connected with sexual or uterine excitement, I have never seen any good from the use of opium. In all these forms of mania, though least markedly in epileptic mania, I have, however, seen benefit from the employment of bromide of potassium, with or without tincture of henbane. In one case, that of a widow of sanguine temperament and active habits, who suffered from acute chattering mania, connected seemingly with uterine excitement, recovery took place within a fortnight under the use of bromide of potassium and henbane. Exactly a year afterwards she had an exactly similar attack, when the same treatment was successful in a short time.

In all those cases of Insanity in which tonics seem to be demanded by the state of the bodily health—and they are the majority of cases, at one period or other of their course—iron and quinine may be given; and one of the best ways of giving them is in a mixture containing quinine, the tincture of the sesquichloride of iron, and chloric ether. In some cases it happens that an uncontrollable diarrhœa sets in and carries the patient off, nothing availing to check it: acetate of lead, with opium, and enemata of starch and laudanum, are most likely to be useful.

When Insanity has become chronic, or when fixed delusions are established, there is small hope of special benefit from drugs. The general health being duly attended to, a systematic moral treatment will be best adapted to restore health of mind. Where there is persistent refusal of food, it must never be allowed to continue so far as to endanger the bodily health; and if persuasion entirely fail, then the stomach pump must be used to administer food, or this may be given by a tube passed through the nostril. Those who are suicidal should be carefully watched at all times, and especially so on getting up in the morning, when the thoughts are gloomy, and the desperate impulse is apt to surprise and overpower them. The monomaniac, who has delusions that he is watched continually, or otherwise persecuted, must always be deemed dangerous to others; for at any time he may become so impatient of his sufferings as to make a fatal attack upon his fancied persecutor. Those who suffer from moral Insanity are often very troublesome to deal with satisfactorily; but it will be worth while always to remember that one unequal to the responsibilities and duties of the social position in which he was born may not on that account be unequal to the relations of a much lower social stratum. It is not because a person insists on ruining himself that it is justifiable to deprive him of liberty by sending him to an asylum.

In all forms of Insanity, it is most necessary to look beyond the mental symptoms, whether these be symptoms of excitement or of depression, to examine closely the physical symptoms co-existing with the mental perversion, and to direct the medical treatment to the nature of the bodily disease which will sometimes be found to be at the root of the whole disorder. Neither opium, nor henbane, nor digitalis, nor any other drug, will act as a specific in any kind of mental derangement; and it is vain to hope and a folly to attempt to get rid of the disease by merely stifling its prominent symptoms. A rational method of treatment must be based on a careful inquiry into the patient's previous history and into the origin of his disease, and on a faithful study of all the symptoms, bodily and mental, which it presents.

HYPOCHONDRIASIS.

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and

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Definition.—A disease of the nervous system, of unknown and possibly varying seat. It is markedly hereditary, being one of the transformed neuroses which descend from a parent stock strongly tainted with insanity. Its principal feature is mental depression, occurring without apparently adequate cause, and taking the shape, either from the first, or very soon, of a conviction in the patient's mind that he is the victim of serious bodily disease. The sufferer's belief in this disease is so firm, that he describes minutely the symptoms which, as he fancies, indicate its existence. But he may place the imaginary malady in almost any organ of the body, and he usually describes some symptoms which are anomalous, or even incredible. Finally, Hypochondriasis may be evoked by a real organic disease acting as an irritant to an hereditarily predisposed nervous system: in this case, the anomalous nervous symptoms may mask, and even conceal, the occurrence of serious changes in some viscus.

Nomenclature.—The same name has been applied to the disease since the days of Hippocrates: it has always been known as "Hypochondriasis," or the "hypochondriac disorder," and sometimes as the "vapours," or the "spleen;" but these last two synonyms are, in fact, mere explanations of the ancient hypothesis which was expressed in the word Hypochondriasis. This hypothesis it is really important to say a few words about, since the vulgar conception of the disease still, though unconsciously, cherishes it; although our improved knowledge of the relation of the nervous system to the organism generally has now made it an anachronism.

The words *ὑποχονδριακὸν πάθος*, applied by Hippocrates and Galen to the disease, imply a belief that the viscera behind the xiphoid cartilage, and below the diaphragm,[1] were its seats; and Galen very distinctly says that *black bile* is its cause. It is worth while to recall for a moment the physiological ideas which Galen, with great ingenuity, had compounded from the speculations of Plato on the one hand, and Hippocrates on the other. According to Galen, the functions of the human body were maintained by three *πνεῦματα* (whence, remotely, our "vapours"). The lowest of these was the *πνεῦμα φυσικόν*, and developed the *natural force* in the liver; the second was the *πνεῦμα ζωτικόν*, which elaborated the *vital force* in the heart; and the third and highest was the *πνεῦμα ψυχικόν*, which developed the *animal* or *soul force* in the brain.[1] Any one who has been curious enough to investigate these questions will see at once that the lowest or "natural" force of Galen is the counterpart of that lowest kind of *mortal soul* which[2] Plato represented as residing in the abdominal organs, and chiefly in the liver, and as having to do with the baser animal passions and the supply of the needs of vegetable life. The ancient tendency to view every source of functional activity as an entity—almost a personality—made it quite consistent for the long succession of Galenist physicians to endow the liver-force with a quasi-consciousness and perception, and even with voluntary activity, though of a low kind; and, on the other hand, the Paracelsian and Helmontian doctrine of the *Archæus* rather added to than changed anything in the extraordinary power over the general organism which was attributed to the abdominal organs. Then the absence of any accurate knowledge of the functions of a central nervous system, the recipient of sensory impressions, and the originator of motor acts, induced men to localize in the various organs the source of the functional disturbances which appeared to be manifested therein. The vagaries of hypochondriacal sensation were therefore, in the ancient view, the perturbations of the natural force generated in the liver, spleen, and pyloric part of the stomach. It is to be remarked, moreover, that Hypochondriasis was very generally confounded with hysteria (to which it doubtless has a relation), quite down to the present century. There is nothing surprising in this. The flatulence which is frequently a striking symptom both in hysteria and Hypochondriasis was, for the ancients, a commotion of the natural spirits which resided in the abdomen.

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[1] The viscera of the hypochondria, to which the ancients attached such importance, seem to have been especially the liver, the *pyloric part* of the stomach, the omentum, the mesentery, and the spleen. The stomach considered as a whole they regarded rather lightly.

Of the long list of authors who have treated of Hypochondriasis, since the days of Galen, there is scarcely one who viewed the disease in any essentially different light from that in which Galen regarded it, until we come to Thomas Willis, the great investigator of the nervous system. It is easy to see what were the common ideas on the subject at the time by consulting that curious book, Burton's "Anatomy of Melancholy," which was published a few years after Willis's death, and which represented the knowledge which a learned and clever layman might pick up without knowing, or without demeaning himself to notice, the writings of a contemporary countryman. Burton says that the general view of authors represents the hypochondriac or windy melancholy as arising "from the bowels, liver, spleen, or membrane called *mesenterium*," and quotes Laurentius as dividing it into three kinds, the *hepatic*, the *splenetic*, and the *mesaraick*.

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[1] De Loc. Affect. v. De Usu Part. v. De Usu Resp. 163, 164.

[2] Timæus, Ed. Stallbaum, §§ 69, 70.

Willis,[1] on the other hand, placed Hypochondriasis amongst the diseases proper to the *diathesis spasmodica*; he made it an affection of the nervous system, but so far conformed to the old ideas as to attribute its ultimate causation to impurity of the splenic blood. In the next century, Flemying[2] ventured a more distinct opinion, that the brain was the part primarily affected; and Cullen[3] and Robert Whytt[4] (especially the former) placed great stress on the share which the nervous system has in the production of the disease. The next prominent step was taken by Georget[5] (1819), who protested against the view (at that time still commonly prevailing) of the abdominal origin of Hypochondriasis, and the practice of powerful purgation, &c. which was based upon it. But the most complete and effective attack on the old view was made by J. Falret,[6] in 1822. This author dwelt strongly on the hereditary character of the disease, and the great frequency with which it is immediately excited by stress of intellectual labour, or by moral and emotional causes. The view of Falret was perhaps carried to excess in limiting the primary seat of the disease so strictly to the brain; but it has prevailed, and Hypochondriasis is now commonly placed among the varieties of insanity. Griesinger, for instance, in his admirable treatise on mental diseases,[7] makes Hypochondriasis a variety of melancholia, which is his first class of "mental diseases characterised by depression;" and Leidesdorf[8] adopts the same view. It will be seen that the view which we hold differs in some degree from this; but there can no longer be any doubt that the true seat of the disease is in the nervous centres.

HISTORY.—The history of a hypochondriac patient is that of his nervous system under the two aspects of its congenital form and the influences—of nutrition, education, and emotion—to which it has been subjected. So far, it is not different from the story of the sufferer from any other form of nervous disease. But Hypochondriasis is distinguished by this,—that a more important part is played by the congenital disposition of the nervous system, and a less important part by the physiological and spiritual influences which have been brought to bear on it, than is the case with the majority of nervous diseases. It comes nearest, in this respect, to insanity on the one hand, and to epilepsy and neuralgia on the other. It is the almost *inevitable* inheritance of a certain percentage of the descendants of any individual who may be very strongly tainted with insanity. On this subject we shall say no more till we can discuss more fully the etiology of this singular disease.

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[1] T. Willis, Opera Omnia, 4to. Genevæ, 1676. The whole treatise, De Morbis Convulsivis, and that on Hysteria and Hypochondriasis (in reply to the strictures of Nat. Highmore), are astonishing efforts of genius, and will well repay perusal in the present day.

[2] Neuropathia; sive de Morb. Hypochond. et Hyster. Ebor. 1744.

[3] Clinical Lectures. London, 1777, pp. 39-57.

[4] Observations on the Causes, Nature, and Cure of the Disorders called Nervous, Hypochondriacal, &c. London, 1777.

[5] De la Phys. du Syst. Nerv. Paris, 1819.

[6] De l'Hypochondrie et du Suicide. Paris, 1822.

[7] Die Path. und Therap. der psych. Krankheiten. 2d Edit. Stuttgart, 1861.

[8] Path. und Therap. der psych. Krankheiten. Erlangen, 1860.

SYMPTOMS.—This phrase is pre-eminently appropriate to the phenomena of Hypochondriasis. Of physical signs we have almost none to guide us; and this is in perfect agreement with the position which this disorder holds in the category of diseases generally. All is in the region of symptoms. For the most part, too, the symptoms are subjective only: still there are features which the experienced physician can detect, and which can hardly be simulated by a malingerer.

The most important external feature of Hypochondriasis is this: that without any sufficient reason for such conduct, and without any signs of intellectual insanity, the patient is observed to concentrate his attention on some particular organ of his body, and to fancy that it is seriously diseased. This concentration of attention is often preceded and accompanied by notable depression or variability of his spirits, with a tendency, on the whole, to depression: this is not always the case, however, for there is sometimes no antecedent symptom connected with the general mental state. In many instances the patient's first sufferings take the form of what he himself considers dyspepsia, but which is in fact little more than flatulence, from the formation of large collections of gas in the stomach and bowels. Along with this flatulence, there are sometimes appearances which give a superficial colour to the idea of a primary stomach derangement: the tongue, for instance, is often pasty and coated, and there may be foul breath; the appetite is not unfrequently ravenous, capricious, or well-nigh lost; there is generally obstinate constipation; in rare cases there are even attacks of vomiting.

More commonly there is an antecedent mental change, the character of which it is at first difficult to seize, and which forms one of the grounds for the modern practice of including Hypochondriasis in the varieties of actual insanity. Before any local symptoms have declared themselves, the patient has already become changed in his disposition: in most cases, perhaps, the change is simply in the direction of despondency or vague alarm, for which he can give no reason. It is remarked by alienists that the mental condition, even thus early, is characterised above all things by an expansion of the *self-feeling*, a pre-occupation of the patient with his own condition, to the exclusion of other interests and affections. This is true; but it appears to us that the self-concentration is more like that of a person in the preliminary stage of an acute inflammation or fever, the nature of which is not yet declared, than the egotistic alteration of character which seems to lie at the basis of insanity, and which probably depends directly upon minute changes taking place in the cortical substance of the brain. It is a real bodily sensation (though at first indescribable) which en-chains the sufferer's attention; and before long this vague feeling is exchanged for a positive localized sense of uneasiness or actual pain.

Sometimes the early mental state is one not merely or chiefly of despondency, but characterised by suspiciousness and irritability of temper, with quick changes from high spirits and loquacity to moody silence. In any case, after a time, the patient not only exhibits in his aspect and conduct the general uneasiness from which he suffers, but begins to complain of definite subjective symptoms. Probably the most common of these is pain of a gnawing or burning character, or else a sense of great though vague uneasiness at the pit of the stomach. But in fact any part of the peripheral distribution of the sensory nerves may be the apparent seat of painful sensation; and besides this there is often a generally heightened sensibility of the skin. Both the active pain and the heightened sensibility of (uncomplicated) Hypochondriasis are subjective, and resemble the similar phenomena which are so common in hysterical women, in vanishing when the patient's attention is powerfully diverted from them. The painful sensations of which hypochondriacs complain are very acute; and their severity concentrates the attention of the sufferer exclusively upon them, increasing the apparent egotism of his disposition. But it may here be remarked, that the heightened self-feeling of Hypochondriasis does not partake of the despondency of true melancholia, still less of the character of other forms of insane egotism. The patient (as observed by Leidesdorf), though depressed in mind, not only wishes to get rid of his malady, but has great faith that he will do so: a faith which suffers repeated shocks, indeed, from the non-success of particular remedies, but quickly revives in favour of some new mode of treatment. The eagerness with which he pursues the means of cure is the true cause of the limitation of his thoughts.

Next to pseudo-dyspeptic symptoms, and the occurrence of pains or anomalous feelings at the pit of the stomach, the most common morbid sensations in Hypochondriasis are, probably, formication of the skin, and *burning pains* in the course of particular superficial nerves. It is noteworthy that, so far as we are aware, the nerve-pains most frequently assume the burning type, rather than the lancinating, throbbing, or aching forms which neuralgia more commonly takes. A common occurrence is the sudden shifting of the pains, or the sense of formication from one part of the body to another, or their sudden extension from a small area which they first affected over almost the whole surface of the body. It is important to distinguish, from these pains, those which occur in the early stages of locomotor ataxy: and, in fact, this may be difficult in some instances, for the ataxic pains also are singularly shifting. However, the pains of ataxy are confined so strictly to the *limbs* (usually to the feet, thighs, and nates), that this of itself constitutes a difference from hypochondriacal pain. Another very frequent subjective symptom is the feeling of pain or great but indescribable uneasiness deep in the heart, or the lungs, or the liver, the bladder, or the rectum. The development of the subjective symptoms is very often seriously influenced by the fact that the patient is driven by his misery to consult medical books, or to pester his medical friends with questions bearing on his sufferings: his defective knowledge and distorted fancy lead him to apply, *à tort et à travers,* the scraps of information which he picks up, and to imagine, successively, that he has discovered in himself the signs of one, two, or half a dozen serious organic diseases. Attention being thus directed to particular organs, the subjective symptoms naturally increase and multiply, and the emotional excitement produced also frequently sets up severe functional disturbance, such as flushings of the face, abdominal pulsation, palpitation of the heart, partial suppression of bile and jaundice, or bilious diarrhœa; symptoms which still further confirm the sufferer in the belief that he is labouring under serious organic disease. A very common delusion is the belief that there is fatal heart-disease; and a scarcely less frequent one is the persuasion of the patient that he is impotent from spermatorrhœa: this last is of course greatly fostered by reading pseudo-medical treatises. In the case of patients whose family is strongly tainted with insanity, the anomalous sensations often assume a type which approaches to hallucination or illusion (as where there is the belief that a serpent is writhing about in the entrails, &c. &c.), or the judgment becomes affected to such a degree that the patient entertains preposterous delusions (as that he is made of glass, and in danger every moment of being broken, that he is being magnetized, that people are conspiring to poison him, &c.). The delusions sometimes are confined, at any rate for a time, to one or two organs, but are nevertheless so extravagant that it would really seem no paradox to say that the patient has a mad stomach, a mad liver, or a mad bladder; while on all other matters his intellect remains healthy, and often unusually acute. It is probable that *any* portion of the nervous centres may be functionally disordered in Hypochondriasis, and thus give rise to disturbances of this kind in the organs with which they are related. But on the subject of the Protean symptoms of Hypochondriasis it is really unnecessary to enlarge further, and we may refer those who desire to read a truthful and highly picturesque description of them to the pages of Burton,[1] to say nothing of more modern writers.

DIAGNOSIS.—The diagnosis of Hypochondriasis, from maladies superficially resembling it, is proverbially beset with difficulties, and the practitioner can only gain confidence in his decision on the more doubtful cases by means of long experience. Nevertheless, the general principles on which his judgment must be formed are not very difficult to state.

If the anomalous character of a patient's subjective symptoms points in the direction of Hypochondriasis, the very first subject of inquiry should be the family history. A well-defined history of insanity in the race would at once indicate the probability that the patient's sensations were partly illusory, and not referrible to their apparent site. On the other hand, a decided history of the absence of insanity and of the other severe neuroses from the family during two or three previous generations would still more strongly suggest that the case was *not* one of Hypochondriasis. The next point for investigation would be the mode of commencement of the illness. A history of the primary occurrence of severe bodily symptoms, whether in the shape of pyrexia, of disturbance of hepatic or gastric functions, or of pain in the course of nerves, is unfavourable to the diagnosis of Hypochondriasis, unless these phenomena were preceded or accompanied by psychical changes such as have been already described. Even a more chronic development of capricious pains, of formication of the skin, of flatulence, palpitations, and the like, is not specially indicative of Hypochondriasis, unless there is unusual anxiety on the patient's part, and an egotistic tendency to dwell on his sufferings. A great deal may be gathered from considerations of age and sex. Hypochondriasis is pre-eminently a disease of adult and middle life; it is hardly ever seen before puberty, and it very rarely makes its first appearance after the age of fifty. It is greatly more common among men than among women; in the latter sex it appears to be replaced, for the most part, by hysteria. Still Hypochondriasis may occur in women, and the question of diagnosis from hysteria, in such a case, becomes important, and may be very difficult. Beyond all other circumstances which favour the probability of Hypochondriasis is the fact of a strong hereditary taint of insanity. The age at which the symptoms commence is important: thus the first appearance of hysterical phenomena nearly always takes place between the ages of fourteen and thirty, or else at the grand climacteric; and has very commonly a marked relation to those changes in the nervous system which correspond with the changes of the sexual apparatus: whereas the development of Hypochondriasis is especially associated with the circumstances of middle life—in the rich and idle with the *tedium vitæ* of a purposeless existence; in the poor and anxious with the cares of a family, and perhaps with the added misery of a conscious failure in efforts to support relations and dependents. Severe moral and emotional shocks may be followed either by hypochondriacal or hysterical disorder; but the latter is the infinitely more probable result in women who are not descended of markedly insane families, and especially in women who lead busy lives.

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[1] Anatomy of Melancholy, pp 270–274.

One of the most important questions in diagnosis is the decision whether, if Hypochondriasis be present, there is not at the same time some organic visceral disease; for it sometimes happens that the first sign of the occurrence of such disease is an outbreak of hypochondriacal symptoms, the patient being hereditarily predisposed to the latter disorder, and his nervous system excited to morbid action by the irritation of the new organic processes which are going on. Of the diseases which have been known to produce such an effect, structural changes of the liver, and, next to them, structural changes of the stomach, are probably the most frequent examples; and, after these, aneurisms of the great vessels, valvular diseases of the heart, angina pectoris. It is unnecessary here, even if there were space, for us to go into the characteristic symptoms of these organic diseases. The first feature which may lead the physician to suspect the existence of organic visceral disease, in the midst of symptoms which he feels sure are hypochondriacal, is the persistence of some one complaint by the patient—*e.g.*, of pain in a particular locality: especially if this be combined, always in the same order, with other symptoms that belong to the suspected organic disorder, and with which the patient is not likely to be acquainted so as to be mentally influenced to reproduce them. Thus if, along with a fixed complaint of pain in the præcordia increased by swallowing, there is the regular occurrence of regurgitation of some of the food very shortly after deglutition, it is a case for inquiry as to the possible existence of cancerous or other stricture of the cardiac end of the stomach, &c. It is needless to say that physical signs, when they are present, are the most valuable helps in discerning organic disease which is masked by Hypochondriasis; yet even here there is need for caution. For instance, the occurrence of hardness and tumidity in the epigastrium or the hypochondrium, in such a form as closely to imitate a scirrhous tumour (even on repeated examinations), may be produced by spasmodic contraction of one or both recti: in such a case the administration of chloroform would at once dismiss the suspicion by dissolving the "phantom" tumour. A circumstance which is always of doubtful interpretation is the occurrence of wasting, especially if combined with jaundice. If this takes place with rapidity, it can hardly be owing to hypochondriacal disturbance of digestion and assimilation, but is probably due either to the generally depraving effect of cancerous or tubercular taint, or to direct interference with nutrition from the mechanical effects of ulcer, stricture, or tumour, upon some of the chylopoietic viscera. The combination of insidious and unsuspected malarial poisoning with hypochondriacal tendencies may produce formidable difficulties in diagnosis, which can only be overcome by careful study of the patient's past history, sometimes by the discovery of enlarged spleen, and above all by the effects of anti-periodic medicines. Another variety of blood-poisoning, which in hypochondriacal patients may be somewhat masked, is chronic alcoholism; but it has been shown in another article in the present volume, that the symptoms of the latter complaint are, after all, tolerably distinct and recognisable from their peculiar grouping, and even in a hypochondriac they may generally be identified.

A more serious difficulty in diagnosis than any which has yet been mentioned is the distinction between certain forms of hypochondriasis and true melancholia. Given a patient with a decided family history of insanity, with a mental condition marked by a strong tendency to dwell on complaints of bodily misery, and with dyspepsia and flatulence, it may be very difficult to say whether or not the case will pass into true melancholic insanity. The following case will give a good idea of the occasional uncertainty. A postman, aged forty-three, a widower, was much overworked, and especially harassed by having to sort the letters in the morning before he started on his beat, a task which had to be hurriedly done, and hence gave him much anxiety lest he should make mistakes. He applied for relief from dyspepsia and flatulence and bilious diarrhœa, but at the same time complained that his spirits were dreadfully low, that he had thoughts of suicide, and that he believed he had "something alive in his inside." A simple tonic mixture of mineral acid and bark, combined with the moral influence of encouraging assurances from the physician, did this patient so much good that he soon seemed perfectly well, and remained so for some months. He then got married again, and his marriage apparently embarrassed his means, though not seriously; but his despondency now returned in the form of a belief that he and his family would have to go to the workhouse (of which there was really not the least danger), and the impulse to suicide again became very urgent. At the same time his dyspepsia and bilious diarrhœa returned. He applied for medical relief, was ordered the same treatment as before, and was encouraged to hope for a speedy cure; but the very next day he attempted suicide by completely severing the whole of his genitals from his body with a razor. He was taken to St. George's Hospital, and with great difficulty kept alive while the wound healed. Six weeks after his discharge from the hospital he appeared before his former attendant, looking fat and fresh-coloured, but more despondent than ever; indeed, plainly insane. He was then lost sight of, but there could be little doubt that he would get worse, and, if not carefully watched, would commit suicide.

Such a case as the above has little to separate it from Hypochondriasis except the one important feature of the early occurrence of *suicidal despondency;* but this feature would probably be sufficient justification for a decided diagnosis. It is only where the hypochondriac patient has been exhausted by a long continuance of his sufferings and rendered hopeless by the failure of a thousand attempts at cure, that he turns his thoughts to self-destruction, and by that time he may be considered really insane. Indeed, the hypochondriac proper regards the idea of suicide with the utmost repulsion and disgust.

There is no serious difficulty in distinguishing simple Hypochondriasis from the other forms of insanity.

PROGNOSIS.—The prognosis of Hypochondriasis varies extremely, not so much with the apparent severity of the symptoms as with the circumstances under which they arose, the length of time during which they have already persisted, and above all the degree to which the patient's family has been tainted with insanity. But in general it may be said that the younger the patient, the shorter the time during which he has suffered, the less that the nutrition of the body has deteriorated, and, above all, the less of decided family taint of insanity that can be traced, the more hopeful is the aspect of the case; and *vice versâ*.

Etiology.—The "causes of Hypochondriasis" is an expression even more singularly unhappy than the average instances of a phraseology of causation applied to those circumstances which precede the outward and visible development of functional disorders. To commence with those influences which have a conventional title to be called "exciting" causes: it is undoubtedly true that in a considerable number of cases the train of disastrous events has seemed to be fired by the moral collapse consequent on over-exhausting labour, or bitter disappointment of cherished hopes, or on the sudden revelation to the mind of an idle man that he is a mere burden on the face of the earth. Again, it is commonly said that reading or conversation on medical topics often frightens laymen, and, more rarely, even doctors, into a nervous and hypochondriacal frame of mind. There is, doubtless, something to be attributed to such influences, but the most thoughtless person could hardly fail to be struck, on reflection, with the surprising infrequency of Hypochondriasis in comparison with the ubiquitous operation of such influences as grief, fatigue, the sense of shameful failure, the habit of miserable and heart-wearying idleness, and the practice by the laity of reading medical treatises. If we turn to the events which would be conventionally spoken of as "predisposing" causes, we are scarcely likely to be more satisfied with the appropriateness of the term "cause;" though we come upon facts of far greater practical value than those which have just been mentioned. The fact of hereditary insane taint, for instance, is an antecedent which is observed in an immense number of cases, if not in all. The preponderance of males among hypochondriacs is equally unmistakeable; and so is the fact that the bulk of cases occur in persons in the prime and vigour of life. It is also an undoubted fact that the average level of intellect in hypochondriacs is not below but rather above the general standard; and that their bodily health has often been excellent up to the moment when the nervous symptoms made their appearance. But instead of saying that these circumstances are "causes" of the disease, it will be convenient to say that they are *conditions* of its occurrence in the following degree and manner. A certain number of the descendants of a family strongly tainted with insanity will invariably be born with peculiarities of the nervous system: these peculiarities do not, probably, consist of recognisable structural faults, but rather of tendencies of one or more (perhaps scattered) portions of the central system to change interstitially in a certain morbid direction, at particular crises of life, through which healthy organisms pass unharmed. Arrived at such crises the nervous system will surmount them, or will succumb, according to the absence or the presence of certain external disadvantages. If the morbid change occurs, it will not affect the machinery of the intellectual and reflective faculties chiefly, perhaps not at all; its force will be spent mainly on that portion of the nervous apparatus which performs the function of translating to the mind the perceptions of sensitive nerves at the periphery: but it is not impossible that even the primary morbid action is occasionally developed in nervous centres which govern secretion and other functions of organic life; and that the dyspepsia, and other functional disorders of viscera, may in these cases be the *direct* result of a central disease, instead of reflex phenomena dependent upon the condition of consciousness, as is probably the case in many instances. In the latter stages of the malady there can be no doubt that the mental depression reacts with great force upon the machinery of organic life, disordering secretions and rhythmic motions very extensively.

The Pathology of Hypochondriasis, in the strict sense of the word, does not exist, for there are no anatomical or physiological facts upon which it can be based. Morbid anatomy has revealed absolutely nothing which in the slightest degree explains the occurrence of the disease, and the physiology of the symptoms is to the last degree obscure and uncertain in its interpretation. It is only in those cases which develop into true insanity, more especially those which pass into dementia, that the brain exhibits any notable changes; and these alterations, when they occur, are no proper part of Hypochondriasis. It is neither impossible nor unlikely that the improved modern methods of examining the nervous centres, if they could be applied to the central ganglia of certain visceral (nerves and especially to the nucleus of the vagus), might detect appreciable changes even in the early stages of the disease. But the opportunities for carefully examining the nervous system of patients in the early periods of Hypochondriasis can rarely be obtained, and it is probable enough that the question as to the pre-occurrence or not of structural changes will never be thoroughly cleared up.

The Treatment of Hypochondriasis consists of the use of moral and constitutional remedies and of remedies for symptoms.

It is obvious that the first duty of the physician is to encourage the hypochondriac to forget his woes; but nothing is so difficult in practice, and that for the best of reasons. It is a fallacy to suppose that the sufferings of the patient are unreal; on the contrary, they are most vividly real, and it is impossible that he should forget them till they cease. Yet the mind has a reflex influence upon the bodily disorder, which may be as effective for good as for evil; and this fact may be taken advantage of. The key to the moral treatment is the breaking down of the patient's morbid self-concentration, and this object may be achieved to some extent in many cases by a change in the course of his daily life. The class of patients with whom this may be most readily carried out are those in whom the constitutional tendency to Hypochondriasis is aggravated by the *ennui* of an idle life: for these an active career or pursuit of almost any kind is an immense gain; only the new occupation should be one which forces them to mix with the world. The isolated activity of the student is no real diversion from the fancies of Hypochondriasis, as the case of

Dr. Johnson, and of many other famous intellectual workers, abundantly proves. It is needless to say that all actively depressing influences should be removed, such as immoderate venereal indulgence, of whatever kind, or alcoholic intemperance. On the other hand, the influence of new emotions which tend to lift the patient out of himself can scarcely fail to be beneficial; and it would be a real good-fortune to a hypochondriac if he could fall in love in a natural and healthy manner, or if he could interest himself warmly in philanthropic schemes or other plans of public usefulness. And, above all, something like a police supervision should be exercised as regards his studies, in order that he may be rigorously kept from the perusal of medical or other books which might remind him of his miseries; for though we do not believe that these things can create Hypochondriasis, yet they can certainly prevent its cure. It is well understood, however, that no good can be effected by simply laughing at his narrative of suffering, or bantering him on his fancifulness; on the contrary, it is necessary for the physician to be interested and to believe in the reality of his painful sensations. If the patient once thinks that the doctor is taking pains to get at the secret of his troubles, he will be inclined to accept the first word of encouragement the latter throws out; and the reflected influence of reviving hope will be certain to assist recovery.

The *constitutional* treatment is to be directed towards improving the general nutrition; and the task here is partly that of aiding the primary process of digestion of food, and partly that of rendering more active the processes of decomposition and exchange in the tissues generally. The hypochondriac either has a deficient, a capricious, or a ravenous appetite, but in any case the primary function of digestion is almost always markedly impaired if the disease has lasted for any length of time; and when this depends on a want of tone mainly, or a condition of irritation of the stomach (such as is indicated by a coated tongue with a red or strawberry tip), the use of vegetable bitters and mineral acids will often do great good. Defective secondary assimilation, which will be especially indicated by the condition of the urine, is generally much benefited by the use of cod-liver oil for a rather prolonged period, if the remedy can only be tolerated by the stomach. In cases where the oil cannot be borne, cream, butter, or some other form of fat, will often agree, and may be made the first stage to inducing the stomach to retain the cod-liver oil. Nor is it by any means only in cases where there is general emaciation that the administration of fat does good; it is probable that the nutrition of the nervous tissues is directly improved by this treatment in many instances. The administration of iron is doubtless of great use to some anæmic patients, and sea-bathing frequently appears to exercise a very beneficial influence: but the first of these remedies is generally most efficacious when taken in the form of the chalybeate waters of some foreign spa; and there is good reason to doubt whether both mineral waters and sea-bathing do not owe most of their apparent power to the moral influences of travel and change of scene and mode of life. The more specific nervous tonics, such as strychnia, quinine, or phosphorus, seem to exercise but a doubtful and accidental influence.

The treatment of symptoms is a thing to be eschewed in Hypochondriasis, with certain special exceptions. While, however, it is desirable to avoid concentrating the patient's attention on parts which are the apparent seat of mere morbid sensations, it is important to relieve him of the distress caused by real (though mere functional) disorders of the digestive system. Decided acidity of the stomach should be counteracted by the use of antidotes, of which none is more efficacious than magnesia ponderosa, in ten-grain doses thrice daily, or Brandish's solution of potash, ten minims three times a day, with gentian or cascarilla. The excessive or too long continued use of alkalies is, of course, to be avoided. The distressing flatulence, which is often one of the earliest, and also one of the most annoying symptoms, is greatly relieved by creasote (one drop in a pill twice or thrice a day), or the infusion of valerian. Alcoholic tinctures should be very cautiously employed, if at all; for there is a real danger of the patient coming to appreciate the comforting sensations given by the spirit so highly, that he gradually takes to drink: this is especially true in the case of hypochondriacal women, as it notoriously is in hysteria. We may add that it is particularly likely to occur in patients exhausted by masturbation, or other venereal indulgence. The constipation, which is frequently so obstinate and troublesome, must be remedied, if it be anyhow possible, without the use of drugs; for it is most dangerous to stimulate the patient's love of self-doctoring in the direction of the habitual use of purgatives. The prescription of fruit, green vegetables, &c., as articles of daily food, is a far more desirable mode of accomplishing our object; and the habitual practice of active bodily exercise is a powerful aid to the same end.

The question of the *quantum et quale* of physical exercise which may be beneficial in Hypochondriasis forms a fitting subject with which to conclude our remarks on treatment, since this is a remedy which directs itself alike to the moral, the constitutional, and the symptomatic condition of the hypochondriac. The only rule, however, which it is possible to lay down for our guidance in this matter, is the direction to employ physical exercise in such a manner and to such an amount as shall fully exercise the muscles without ever producing severe fatigue, and shall also be amusing to the patient. It is a very dangerous error to carry exercise to the fatigue point; a short continuance of such malpractice will usually suffice to produce a profound deterioration of the vigour of the nervous system, and an aggravation of the hypochondriacal fancies.

HYSTERIA.

By J. Russell Reynolds, M.D., F.R.S.

The most characteristic feature of Hysteria has been held to be a particular form of convulsion, which will be described hereafter in detail; but there are many phenomena, some falling far short of convulsion, even when they resemble it in kind, and others differing widely from it in their form, and these have been termed "hysterical." If, therefore, by the word "Hysteria" is intended a definite disease, the term "hysterical" should be employed with a no less definite aim; but it has unfortunately been sometimes used to denote either a mere variety of the "nervous temperament," a mood or disposition of the health of both body and mind, or sometimes a vague condition of disordered function which cannot be conveniently placed elsewhere. The employment of the word "hysterical" may sometimes be found indicative of the state of the mind of the practitioner rather than of that of the patient's health. It simply conveys a doubt as to what is the matter, but expresses a prevailing conviction that it is nothing very serious as to life, and that it might culminate in an attack of convulsions of the kind called "hysteric."

The vast preponderance of Hysteria in the female sex has given rise to its name, and to a theory as to its essential nature and mode of production. It has, however, and especially of late years, been so distinctly shown that Hysteria may exist among men, that the older nomenclature, although retained on account of its practical utility, is virtually exploded as to its etymology. Hysteria is not necessarily associated with disease or derangement of the generative organs of either sex: such association may and does very commonly exist; but the true nature of the malady may be overlooked if regard be paid exclusively to that particular relation.

The hysterical condition may exist for many years, and yet be unattended by any distinct convulsive paroxysm. The latter never exists without the former. There are at the present time numberless individuals whose lives are, to themselves and their friends, the sources of more or less constant misery, from the fact of their being distinctly and definitely hysterical, but in whose history there has never occurred a single attack of characteristic convulsion. We cannot, therefore, draw the line of definition so as to include the paroxysm and exclude all beside; but must, on the other hand, regard as essential that which is constantly present, and recognise as the disease Hysteria a peculiar condition of the nervous system of which the paroxysm is only one, although a highly important feature. The mental state of the hysteric patient is more constantly and as characteristically altered as is the condition of the muscular system. There is a defective or perverted will; an increased activity of emotion, and sometimes of thought; an altered and augmented general sensibility; an exaggeration of all forms of involuntary motility—ideational, emotional, sensational, and reflex; and usually some distinct perversion of the physical health. It is almost impossible to frame an accurate "definition" of the disease; and it seems to me more desirable to make the above general statement than to attempt a less detailed description for the purpose of giving it the form of a definition.

NATURAL HISTORY.—I. CAUSES.—*Sex.* Doubtless the most frequently predisposing cause is that condition of the nervous system which is more or less characteristic of the female sex. Hysteric women are met with daily; hysteric men and boys are of comparatively rare occurrence. When Hysteria is found in either a man or a boy, it is to be observed that such person is, either mentally or morally, of feminine constitution, or that he has been overworked mentally, exposed to much emotional disturbance, or greatly reduced in physical power. It is well known that men have frequently become hysterical as the result of some severe accident, such as a fall from a horse or a collision in a railway train. The predisposition to Hysteria does not exist in the fact of an individual having the organs of one or the other sex, but in the possession of a nervous state—an habitual, constitutional, or induced relation between the several elements of mental, moral, and physical life—which is common to, but not always possessed by women; unnatural to, but sometimes exhibited by men. Some women are as little likely to become hysterical as some men are to fall pregnant; they are of masculine build, both mentally and bodily, and their existence and their predispositions to disease furnish another proof of the truth of the general proposition, that it is in the nervous endowments, and not in the nature of the reproductive apparatus, that the special predisposition lies.

Age.—In the female sex, Hysteria usually commences at or about the time of puberty, *i.e.* between twelve and eighteen years of age; but, when once developed, the symptoms may remain throughout life. At the climacteric period Hysteria may become developed in a previously healthy person;—this is, however, comparatively speaking, rare: but examples of extreme Hysteria may be met with after the climacteric period has been passed—and that without unusual discomfort—in some women who were hysterical in earlier life, but whose hysterical symptoms were then less pronounced. Of 351 cases analysed by

Landouzy,[1] the ages at commencement were distributed as follows :—

From 10 years to 15 years	48 cases.
,, 16 ,, 20 ,,	105 ,,
,, 21 ,, 25 ,,	80 ,,
,, 26 ,, 30 ,,	40 ,,
,, 31 ,, 35 ,,	38 ,,
,, 36 ,, 40 ,,	15 ,,
Above 40 years of age	25 ,,
	351

Boys sometimes exhibit hysteric symptoms at puberty, but the most marked cases of the disease that I have seen in males have been at a more advanced age, viz. from thirty-five to fifty and upwards. It will be seen, therefore, that Hysteria may occur at any age ; and it must be remembered that many of the symptoms of senile decay, and many of those which accompany chronic degenerative disease of the brain in middle life, are often called "hysterical," because they correspond very exactly with those which, when they occur in earlier years, are described, and without hesitation, by that word. As decaying life passes on to a "second infancy," we see the signs of a "second childishness :" but often, before either of these terms would be strictly applicable, we may observe something that resembles, in its nervous characters, the period of commencing puberty ; for often the first sad signal of faltering power is either undue emotional disturbance or deficient control of its display. We call these states "hysterical," and may perhaps sometimes use that word very loosely ; but the resemblances are real, and may help us to appreciate the pathology of the disease "Hysteria."

Temperament.—If by nervous temperament be meant simply an hysterical condition, it is unnecessary, and would be wrong, to speak of the one as a predisponent to the other. The two expressions are sometimes used interchangeably; but this is a great mistake: the truly nervous temperament implies no disproportion, *inter se*, of the several nervous endowments; all are alike active. There may be in those of nervous temperament a higher but a shorter life ; an intense vitality, which burns itself out before its time : but the hysteric condition is essentially one of disproportion, and it is not encountered with any especial frequency in those who have previously exhibited the exaltation described. This latter is peculiar to some individuals, but is not necessarily morbid.

Sexual Condition.—Undoubtedly Hysteria is more common in the single than in the married, but it is not limited to the former, and it may exist to its highest degree in the latter. It is said that the wives of incompetent husbands, and barren women, as well as widows and old maids, are frequent victims of the hysteric malady ; but statements such as these do not convey the whole truth in regard of the etiology of Hysteria, and it must be remembered that Hysteria is met with in those who are happily married, in pregnant women, and in nursing mothers.[1] From what has already been said in regard of the age at which the disease begins, it is obvious that age, rather than sexual condition, is the predisposing cause. Hysteria commences at a time of life when girls are, in this country at least, held to be too young for marriage. The fact of its existence, to a high degree at any rate, does not increase their social chances in this particular; they often remain single because they are hysteric, and this probably quite as often as that they become hysteric because they are single. Such a condition does not tend to improve itself, and the disappointment of being left alone may keep up and exaggerate the morbid state. The facts that there are to show that marriage has sometimes cured the malady are not so numerous, nor are they of such character, as to outweigh the evidence to be derived from the persistence, and even aggravation, of Hysteria after marriage. Moreover, it is well known that hysterical women, who are married, are often frigid, and averse to sexual intercourse, and that their aversion is not necessarily due to pain. It is comparatively easy to shrug the shoulders and utter innuendoes over a case which baffles treatment by well-directed regimen and medicine; but does it not sometimes happen that such hints are only a clumsy excuse for the failure of therapeutics? That marriage may be of use in Hysteria is not denied, but it is asserted that it may be so by other than its mere sexual relationships; there may be a number of circumstances which are changed by taking this step in life—annoyances may be removed, new purposes conferred, work given to do, and strong help rendered kindly for the doing it; and all these may concur to lift the hysteric woman out of her former self. If, then, we are regarding Hysteria etiologically, all these points should be considered, and the argument from the effects of marriage should not be based upon one element alone.

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[1] Traité complet de l'Hystérie, Paris, 1846, p. 184.

Sexual excesses are held to be sometimes productive of Hysteria; and doubtless they are so occasionally in men, but there is a deficiency of evidence to prove this in regard of women. According to Duchatalet, Hysteria does not exist with especial frequency in women of the town, and in those who are exposed to excesses of this kind.

Temperature, Climate, and Season.—There are many facts to show that warmth of climate and the seasons of spring and summer conduce to a production of the hysteric condition, but it has yet to be shown what is the element comprised under those terms which is of etiologic moment.

Occupation.—It is demonstrable that absence of employment, as it is commonly met with among the upper classes, favours the production of Hysteria in women; and it is equally clear that overwork, anxiety, and great "strain" upon the intellectual and moral faculties, lead to the development of Hysteria in man. It is, however, questionable whether the mere fact of occupation, or its kind, is either favourable or unfavourable to health or to Hysteria. The unoccupied life of woman is one of exposure to numberless disturbing causes, as is also the over-occupied life of man. The woman, or the young girl, who has nothing to do—nothing serious to which her mind is turned—finds "time" to be egregiously tedious; and she has to choose between two evils, either that of "doing nothing" with it, or that of "getting rid of it" by utterly frivolous pursuits, the distraction of reading silly books and doing the sillier things that "society" prescribes. She thus brings upon herself all the petty annoyances of selfishness and wounded pride, and all the tease and turmoil of unreal and extravagant emotions. The man who is "overworked" finds, commonly, in that work itself, and in that which leads to it, much more than mere mental occupation, viz. anxiety, suspense, and worry, with their concomitants, loss of both appetite and rest; and it is these which, by the nature of their operation, develop the hysteric condition, rather than the amount of simple work which has to be undertaken or pursued. It is then, I believe, neither the presence nor absence of occupation, *per se*, which conduces to the development of Hysteria in either sex; but in the one sex the "nonsense" that takes the place of sober work, and, in the other, the emotional disturbance that attends upon over-exertion. These lead, under apparently dissimilar circumstances, but in a really analogous manner, to the production of the same result. That which is common to the two conditions is an excess of emotional disturbance; but in the one case it is the outcome of idleness, in the other it is often the cause and motive of overwork. Absence of occupation may give time and opportunity for the manufacture of feelings that are tormenting and unreal; over-exertion may be the means taken to relieve the anxiety and emotion which are already pressing. Either condition may be met with in either sex, but the common distribution of work and idleness in relation to Hysteria is that which is indicated above.

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[1] *Vide* Niemeyer, Handbuch, p. 356; Hasse, in Virchow's Handbuch, p. 212.

Menstruation.—It has already been said that Hysteria breaks out most frequently at or about the time of the commencement of puberty; but it has not yet been shown that it has any definite relation to the varying conditions of menstruation. In an individual already hysterical there is or may be more than usual disturbance at or near the monthly "periods," and this is exaggerated by any kind of irregularity which may exist. Menorrhagia, by reducing the vital power, through loss of blood; dysmenorrhœa, by effecting the same result through nervous exhaustion; or amenorrhœa, by its physically direct, and mentally and morally indirect influence, may, either of them, conduce to the increase, or even development, of the hysteric state: but it has yet to be shown that either one of these is of itself sufficient to produce the disease. For it must be remembered that Hysteria may exist to its highest degree,—it commonly has done so in the cases which have fallen under my own observation,—in individuals who have presented no anomaly whatever in regard of the menstrual functions; and, yet further, that it is found in the male sex, which may be influenced much by sexual conditions, but which exhibits nothing analogous to those perturbations that have been mentioned. All that I can add to the above is, that I have found no one condition, either of excess, defect, or perversion of the menstrual function, so commonly or prevailingly associated with Hysteria as to give to it any special claim to consideration in the etiology of the disease. Some people become more or less hysterical about everything that is wrong in the performance of their functions: it is both obvious and notorious that the uterine functions are invested in the minds of women with an amount of importance that is more than their physiological due; and hence it is that menstrual derangements are regarded by them as exerting much more influence than those of the digestive or the excretive functions, and are therefore brought prominently forward in the statement of their cases. That they do exert this excessive influence may be due partly to their intrinsic nature, but partly also to the results of thought and feeling about them; and it is important to bear in mind, for etiologic purposes, this latter element in their mode of action.

Hereditary taint has not been shown to exert any marked influence in the development of Hysteria. It is well known that members of one family occasionally exhibit similar symptoms, but then it must be remembered that many conditions besides those of blood-relationship are ordinarily common to the individuals of one family; they may share equally in what is good or bad in respect of example, education, and surrounding circumstances. M. Briquet states that those who are born of hysterical parents are twelve times more liable than others to the outbreak of Hysteria. Very different numerical statements are made by others, and we must remember that besides the direct transmission of an hereditary taint there is much contingent upon having an hysterical mother.

The most frequent *determining cause* of an outbreak of hysteric symptoms is some mental or moral disturbance; either a violent and unexpected commotion, or more commonly the occurrence of a trivial circumstance which takes the individual by surprise, overcomes the power of restraint, and gives evidence of what is often an ill-understood, but long concealed, annoyance or distress. Sometimes the determining cause is physical, *e.g.* an accident, a loss of blood, or an attack of acute bodily illness, such as one of the exanthemata, pneumonia, or pleurisy. It then appears, and sometimes to the surprise of even the most intimate relatives, that there is a morbid condition of both mind and body which is difficult to describe, and often much more difficult to manage. There may be the extreme symptoms of the hysteric paroxysm, or some of the marked features of the hysteric state. These have now to be described.

II. SYMPTOMS.—It is convenient to separate the paroxysmal symptoms from those which exist in the intervals of attack; and the former will be best understood by those who have become acquainted with the latter, for the hysteric state does much to explain the hysteric paroxysm.

It will be well to divide the interparoxysmal symptoms into several categories, describing separately the condition of the mental and emotional, the sensorial, motorial, and general health.

(*a*) INTERPAROXYSMAL SYMPTOMS.—*Mental and Emotional State.*—The will is perverted and defective, while ideas and emotions exhibit excessive activity. The patient says that she cannot do this, or cannot bear that; and, while under the belief that these things are impossible, they are so. It is often obvious to others that no physical impossibility exists; but it must not be supposed, therefore, that the patient is pretending or "acting a part." For the time being it is often true that the hysteric patient states the fact. What she wants is motive, and this may be sometimes supplied by a sudden alarm, or by an accidental circumstance; but under ordinary conditions the motive is wanting, or is held in abeyance by some imperious idea or emotion. There is an exaggeration and perpetuation of what exists in all people under certain circumstances. Fear paralyses the strong man, while sudden alarm may make the weak man strong. A prevailing idea may limit as well as induce movements which the will can neither counterfeit nor hinder. Let this be remembered, therefore, at the outset in describing the symptoms of Hysteria.

The patient asserts that she cannot control her thoughts, emotions, expressions, or general movements; that she cannot move this or the other limb; cannot open the eyes; cannot stand or walk; cannot relax the rigid spasm of the hand or of the leg; and what she says is true under the existing conditions. But often, under the influence of some unexpected idea, or emotion, or sensation, she does the very things that were said to be impossible.

A patient may be carried into the room, and may fall when left for a moment to herself; tell her to walk, and a wooden doll seems as capable of movement; but, under the stimulus of a wish that what she is saying should not be overheard, she walks to the open door and closes it. Certain ideas seem rampant in her mind; she cries about them, and gesticulates in the wildest manner: tell her to be silent, to keep them to herself, or to control her feelings, and you find them exaggerated, and she affirms that "all the world shall hear" what she has to say; but a gentle rap at the door, that may come from the hand of some one from whom she wishes to conceal her state, is sufficient in a moment to hush this stormy talk, to compose her face, to dry her eyes, and make her speak and smile with placid composure. Sometimes she speaks in a whisper only, and if asked to "exert herself," or "make an effort," so that some particular friend who is a little deaf may hear what she has to say, the only effect is that the whisper becomes quite inaudible, that she makes less sound than ever, and often none at all—she moves her lips, but not even the ghost of a sound is heard to pass them; and yet this self-same person may, when no attention is directed to the voice, speak loudly enough to be heard and understood in the adjoining room. The fact seems to be that the will can be called into exercise only by some one dominant idea or emotion; and that it is this which determines the varying phases of the mental state. Under its influence the hysteric patient may submit to pain, annoyance, and privation such as a healthy person would shrink from without concealment; and under its influence, also, she may be unable to do what any one else could accomplish without either difficulty or fatigue.

Ideation is often excessively active in regard of certain classes of thought; there is sometimes quite a preternaturally acute condition of intelligence in certain directions, *i.e.* in those directions wherein lie the morbid notions which are at the foundation of the malady. Often the hysteric patient makes many mistakes, and attributes to people and circumstances motives and meanings which they do not possess; but very often she exhibits marvellous ingenuity in perceiving the ideas of others, and in unravelling the intention of complicated conditions, when these have happened in any way, or to any degree, to have had relation to herself. Apart from these direct personal relationships the mind often is, or seems to be, a perfect blank; the patient is listless, apathetic, and dull; a most uninteresting specimen of humanity; becoming of value only when her vagaries afford curious illustrations of certain pathological laws. There is a prevailing belief in the importance of self, and the patient thinks that she differs from every other human being; that ordinary laws do not apply to her; that she is "not understood," as the phrase is; and that only some very *outré* or utterly novel mode of treatment can do her any good. She believes all this, and acts upon it with a pertinacity "worthy of a better cause," exhibiting as much energy of purpose in a wrong direction as would serve to cure her were it rightly ordered.

Emotion is commonly excessive in itself, and also in its expression. The patient is hurried from one extreme to the other with almost ludicrous rapidity; and often she walks, as it were, constantly upon that narrow line where tears and laughter meet. Laughter and sobbing not only alternate, but coexist; and often without any obvious and sufficient reason for either. There is sometimes listless indifference to everything of ordinary interest; sometimes absorption in some trivial object; often great restlessness and impatience, with extreme irritability of temper on any attempt being made at control, or any suggestion being offered of change.

These mental and emotional conditions are liable to much variation. Sometimes the patient exhibits them for a day or two, and then becomes like an ordinary mortal; sometimes they are persistent, and vary only in the degree of their intensity.

In Hysteria we occasionally meet with somnambulism, cataleptic conditions, and syncopal attacks, which latter sometimes pass into the state of "trance," or apparent death, of which several cases are on record. These are, however, among the rarer features of the malady.

Sensorial Condition.—An exaltation of sensibility generally may be the earliest, and sometimes the only sign of the hysterical condition. It may, and more commonly does, exist in association with other symptoms, or in the intervals of their recurrence. But sensibility may be altered in several distinct directions; there may be increased, painful, perverted, or diminished sensation, or there may be absolute though partial anæsthesia and analgesia.

Hyperæsthesia is very commonly confounded—nominally and perhaps theoretically—with painful sensibility or sensation; from which, however, it is quite distinct. The hysterical patient often exhibits true hyperæsthesia; she does actually see, hear, smell, and taste what would not be perceptible to those in health, and to herself at other times. The exaggeration of hearing power is that which is the most frequently observed; hysteric girls do sometimes seem to "hear through stone walls:" they detect the minutest change in odour or in taste, and exhibit an exalted keenness of sight. Their sense of touch is also sometimes preternaturally acute.

Painful sensation, or dysæsthesia, is almost always present to some degree. One patient cannot bear the light, another is distracted by the slightest sound, to a third all ordinary odours are intolerable, and to others certain tastes, or the contact of sundry innocent articles of clothing, are most highly objectionable. Pain on pressure of the skin is very common; and sometimes this symptom is universal, but more commonly it is limited in its distribution, *e.g.* to the occiput or the spinal column. It is to be observed that the direction of attention to them always makes the painful sensations much more intense; the mere fact of asking a question about them may sometimes develop them. A middle-aged hysteric woman, whom I saw in hospital a few days ago, had been lying for weeks with her hand before her eyes "to keep out the light" of a dull London sky. Bringing a candle before her—the room being so dark from an accidental fog that I could not see the pupils—she shuddered, knit her brows, and held both hands between her and its feeble light. There was no undue contraction of the pupils, and when her mind was distracted to the condition of her front teeth—the light being still close to her eyes—the brows were relaxed, the hands removed, and there was no expression whatever of uneasiness. This is but one example of a large class of dysæsthesiæ which may be commonly observed in the hysteric.

A lady to whom I was speaking lately, in a tone by no means loud, exclaimed in a voice much noisier than mine, and putting her hands to her ears at the time, "Not so loud—not so loud;" but, a moment afterwards, she stirred the fire so vehemently, and made so much noise in the process, that it was positively annoying to myself, and this without appearing to give herself the least uncomfortable sensation. Sometimes there is obvious and distinct painfulness of sensation upon impressions of ordinary intensity, and this may be limited to one or another organ. Most commonly, however, the pain is not necessarily associated with the impression, is of variable kind and locality, and intermittent in its form.

Besides these alterations of sensibility, hysteric patients constantly complain of "pain," more or less spontaneous in its development. Such pain, wherever it may be situated, usually requires several strong adjectives for its description, and the account given of it is sometimes tediously minute. I have heard one hysteric lady enumerate and detail nine different kinds of pain in her chest! Of these some were bearable, some "intolerable," others "agonising;" and four or five of them usually appeared together, and were present at the moment of description,—and yet the face was calm, and simply conveyed the expression of interest in the description

The localities of pain are very numerous, but some are much more frequently complained of than are others. The favourite haunts of hysteric pain are the top of the head, the left mammary region, the hypogastric, and the sacral. Sometimes the coccyx, and often one of the joints of the limbs, is fixed upon; but I have rarely observed any definite pain in the reproductive organs. When one of the joints is painful, and there is much tenderness of the affected part, it will commonly be found that passive movements or even succussion of the limb may be borne without complaint, whereas even gentle pressure of the skin is described as "agonising in the extreme."

There are other sensations of which much is heard that do not fall into any of the foregoing categories. The processes of ordinary life, which in health are unfelt, become sensible, and sometimes painfully so, to the hysteric patient. She feels the want of breath, the action of the heart, the intestinal movements, the processes of micturition and defecation, and those of sexual intercourse to an exaggerated degree, and in a distressing manner. She feels "short of breath," although there is no actual acceleration of the respiratory movements; "palpitation," although the action of the heart is normal; rolling and rumbling movements, when such are not perceptible to the physician; and distress or difficulty in relieving either the bladder or the rectum, when there is no physical disturbance of such processes. Sexual intercourse is sometimes quite impossible from some morbid sensitiveness of the vulva or spasmodic action of the sphincter vaginæ, neither of which can be detected on examination by the medical attendant.

There is, further, the sensation termed "globus hystericus," which is often, but by no means universally present. It is not always of the same character. Sometimes it is the feeling of a "great lump" in the hypogastric region, rising through the epigastrium and chest, to the throat; but this is, so far as I have seen, extremely rare, and is more frequently met with in books than in practice. The commonest form of distress is that of a ball or lump in the throat; a something which the patient cannot swallow, and which she feels will "choke" her. Anything round the neck is intolerable; she feels as if "something were tight there," although there may be nothing present in the form of dress; she makes constant attempts to swallow, but the "lump will not move;" and these discomforts are exaggerated by sobbing efforts which very frequently attend them. Sometimes the patient puts her finger in her throat to cause vomiting, that she may "bring it up;" sometimes she drinks largely to "push it down;" but in spite of both classes of effort the lump remains. Children when about to cry know what the feeling is, and probably it is of the same nature in the adult.

Diminished sensibility, and even actual anæsthesia of certain localities, may also be found. Anæsthesia may exist over the whole or a large portion of the surface of the body, and may extend to the deeper tissues, to the muscles,[1] and even to the nerves of special sense. It most commonly follows an hysteric paroxysm, but it may be met with when no such seizure has taken place. Commonly it is limited to certain parts, such as the back of the hand or foot, but it may be paraplegic or hemiplegic in its distribution, or may be found along the course of one or more intercostal nerves, and in the mucous membrane of the nose or mouth. The temperature of anæsthetic limbs is sometimes reduced. It is a rare event in any locality, but it has existed in several cases that I have seen in the mucous membrane of the vulva and vagina; the anæsthesia in these instances having been absolute, and that in highly hysterical, married women, who had borne and were still bearing children. Probably, anæsthesia is the cause of occasional retention of the urine and of the fæces in hysterical patients, as either bladder or rectum may be found sometimes enormously distended without the patient being aware of any other discomfort than that occasioned by the swelling. Loss of the senses of sight, hearing, smell, and taste also sometimes occur as phases of hysteric anæsthesia.

Motorial Condition.—In health there are different kinds of movements which the muscular system exhibits; some are voluntary, others depend upon idea, a third group upon emotion, a fourth upon sensation, and a fifth upon impressions which are not felt. There is, moreover, a certain relation between these which is characteristic of health, but either of them may be so altered as to disturb the balance of well-being, and constitute or exhibit either derangement or disease. In Hysteria the normal relation is perverted, and there is an excess of the involuntary motility, and a diminution of the volitional; the former overrides the latter: and not only so, but the particular elements of the former display their powers in an altered ratio; sensational movements are not in due relation to emotional, nor are the latter to those of idea. Generally speaking, the lower acquires the ascendency, and exhibits the most marked phenomena.

Involuntary muscular activity may be increased in regard of either the readiness with which it is called into exercise, or the force and persistence of its display. If these involuntary movements be placed in the following order,—ideational, emotional, sensational, reflective and organic,—it will be found generally true that the increased readiness of action is found at its maximum in the first, whereas augmented force and persistence of action are observed most distinctly in the last; and that the two kinds of alteration diminish in passing from either one of these extremes towards the other.

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[1] See article, Muscular Anæsthesia.

Voluntary movements are sluggish, the patient often lying about on sofas, or in bed, and saying that she is quite incapable of doing this or that. Irrational movements are in excess; and, under the influence of morbid "notions," gestures may be assumed, and sundry physical efforts performed which transcend the powers of health. All the emotional movements are exaggerated; the individual laughs, cries, and makes grimaces of the most distressing kind, and manages often to do that which she could not do under ordinary circumstances.

Sensational movements are in excess: the brows are knit and the eyelids contracted upon the approach of light; there is starting and tremor upon the occurrence of any sudden sound or jar; there is violent spasm upon the production of any pain; and this often without any real exaggeration of sensibility. Reflex motility is greatly increased and perverted, so that spasms arise from "irritations" which in health would be passed by unnoticed; and numberless movements, of momentary duration and varied locality, occur in relation to impressions which are quite inadequate to produce them in the normal state. Other muscular actions, tonic or persistent, exist in various parts of the body, which differ from the reflex in the fact of their permanence, and must be referred to some induced change in the nervous centres. Of these, histrionic spasm, cramps, and long-continued spasms in the limbs or elsewhere, afford the most frequent examples. These tonic spasms are not confined to the muscles of the limbs, they may affect the pharynx, the œsophagus, the stomach, or the intestines; causing difficulty of swallowing or breathing, vomiting movements, strange noises, eructations, hiccup, borborygmi, and griping pains.

But besides the alterations mentioned, there is another kind, which is less common, but of more serious character, viz. paralysis. Loss of power usually occurs as the sequel of an hysteric paroxysm, but it may sometimes take place spontaneously.

M. Landouzy[1] states that, in 47 cases of hysteric paralysis, the distribution of the symptoms was as follows:—

General paralysis of motion and sensation	3
,, ,, of sensation	2
Complete left hemiplegia	8
Complete hemiplegia, side being doubtful	6
Paraplegia	9
Partial paralysis	19

Usually only one extremity is involved, sometimes only a part of the limb; but in many instances there may be what is roughly termed

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[1] Traité complet de l'Hystérie, p. 106.

"hemiplegia." In the latter case the face and tongue are rarely affected, and the paralysis is incomplete; and the motion of the partly paralysed leg is not like that of an ordinarily hemiplegic individual. Dr. Todd says that the patient "drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no act of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks."[1] This is true of many cases. And there is a yet further condition which may be noticed, viz. the absence of any special paralysis of the extensor muscles of the toes. If an ordinary hemiplegic patient be made to walk, it is seen that on attempting to raise the foot from the ground the toes droop and the leg is circumducted; but the hysteric patient does what the healthy person cannot help doing, except by a strong effort of the will,—when making the attempt to walk, she causes an elevation of the great toe at the time of endeavouring to move the foot forwards. The paralytic patient looks at his feet, the hysteric patient looks at her observers. The electric irritability persists in the palsied limb, and its nutrition does not become affected; but there is sometimes diminished sensibility to the electric current; it is at other times normal, and in some cases notably increased. Occasionally the electric contractility and sensibility are both diminished or abolished. Paraplegia is a form of malady sometimes witnessed, and patients suffering in such manner may keep their beds, or leave them only to exhibit a most curious mode of progression, or a series of falls which are quite unlike those arising from organic lesion of the spinal cord. At this time I have under my care two cases of hysterical paraplegia, in which the symptoms are almost precisely similar. The limbs are well nourished, there is perfect electric contractility and sensibility; the patients when lying in bed can elevate their limbs, separately or together, to any height that is required; they can move all the toes, and cutaneous sensibility is intact: but if they attempt to walk, their legs appear to be no stronger than pieces of wet paper, and they tumble down and bruise themselves in various quarters. What, however, is peculiar in the attempt at walking is this, that no amount of help, such as a strong arm on either side, prevents the staggering and falling, but the patients tumble down to within a few inches of the ground, and then recover themselves without help. An ataxic patient would walk, comparatively speaking, well with such assistance; a really paraplegic patient could not so recover the upright position. Where paraplegia has been due to Hysteria, I have not found loss of power over the expulsors or the sphincters of either rectum or bladder; although it often happens that the latter exists without the former. General paralysis is extremely rare, and is only imperfect in degree.

Vaso-Motor Condition.—Sometimes hysteric patients, after a slight rigor, exhibit a certain amount of fever, with headache, and mild delirium, which speedily pass away, or give place to paralysis of the kind described. An irregular distribution of temperature, or the fitful occurrence of sweating, of salivation, or of increase in some other secretion, afford further illustrations of disturbance in that portion of the nervous system which is related to the control of blood-supply.

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[1] Clinical Lectures, p. 620.

The *General* symptoms in Hysteria, or those outside the nervous system, are not distinctive in their character. Usually the patient is not in robust health; there is some pallor, and failure of nutrition; or there may be a great tendency to deposit of adipose tissue. Sometimes there is a condition of habitual ill-health, or delicacy; digestion is impaired, or the uterine functions are irregular, or there is some constant, but more or less indefinite, grievance in the head, thorax, or abdomen. On the other hand, there are many cases of Hysteria in which the general health is good; the patient eats, drinks, sleeps, feels, and is "well."

Numbers complain of nausea, and eructations, or vomiting; but in many cases these symptoms have been entirely due to errors, and even absurdities of diet, and in not a few to excess of stimulants. It is by no means rare for hysterical people to "take to drinking." Alcohol relieves them for a time; is often recommended by medical advisers; the patients know its power to diminish their passing discomforts, and push, beyond all reasonable bounds, their recourse to its aid. Flatulence and borborygmi of the intestines are common enough; and so are palpitation of the heart, syncopal feelings, and dyspnœa—the last, however, without any notable change in the ratio of respiration to pulse. Large quantities of pale, limpid urine, of low specific gravity, are passed; but this is also true of epilepsy and of many other diseases of the nervous system. With regard to affections of the generative organs, there is great discrepancy of opinion. Landouzy states that in twenty-six, of twenty-seven cases, there was some abnormal condition;[1] and, further, that of sixty-seven cases of Hysteria, the symptoms coincided with material alterations of the generative apparatus in fifty-eight, and that in nineteen cases the Hysteria was cured after the removal of the genital affection:[2] but it is, so far as my experience extends, the exception and not the rule to find any definite malady, or indeed definite complaint, in this direction; while in a vast number of cases there has been absolute health in all particulars relating to the reproductive organs.[3]

When men have presented hysterical symptoms, there have always been, in my practice, considerable deterioration of the general health, an impaired nutrition, and a feeble circulation, with exhausted brain.

(*b*) PAROXYSMAL SYMPTOMS.—The attacks of hysteric convulsion do not pass through stages that can be defined, like those of epilepsy, to which they sometimes bear a rude resemblance. They differ widely in degree of intensity, but have a general similitude *inter se*, and can rarely be mistaken in either their slighter or severer form. When slight, they are but an exaggeration of the interparoxysmal state; when severe, they have been confounded with certain grave diseases.

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[1] Traité, p. 171.

[2] Op. cit. p. 174.

[3] Niemeyer says: "Unter den Krankheiten des Uterus sind es ausser dem Infarct namentlich die Geschwüre des Muttermundes und vor Allem die Knickungen der Gebärmutter, welche am Häufigsten zu H. führen, wahrend bei bosartigen Neubildungen und bei destructiven Prozessen hysterische Zufälle weit seltener sind."—*Handbuch*, 2er Band, p. 356.

The following may be regarded as the description of a severe attack. A patient is talking vehemently, often unreasonably, and is agitated in manner; she is crying or laughing, or both, and perhaps apologizing for or lamenting her weakness; friends are either scolding or condoling, and sometimes there is a combination of both modes of domestic treatment; some real or imaginary grievance is uppermost in the mind and the conversation, and is not "met" or removed by the endeavours of the friends. Suddenly the patient gives a scream, or makes a spluttering noise, appears to lose voluntary power and self-control; she falls down with snorting breathing, and a quasi-tonic contraction of the muscles of the extremities and the trunk. She makes hideous grimaces and outrageous noises, throws her limbs about in a disorderly manner, utters incoherent sentences, adopts histrionic attitudes; complains of her throat and stomach, and breathing; appears exhausted, or faint, and sometimes stupefied; occasionally she seems to lose her consciousness, and then, after a fit of "crying," to be "herself again." The whole paroxysm may last for a few moments only, but more commonly it is of much longer duration; a number of absurd gesticulations and irregular convulsive movements lasting from a few minutes to three or four hours, after which the patient seems worn out, and falls asleep.

These points may be observed during the attack :—There is rarely absolute or sudden loss of consciousness; the patient does not fall in such manner as to hurt herself, or tear her clothes; there is somebody near who shall see the phenomenon; hysteric paroxysms do not occur during sleep, or when the patient is alone; there is something artistic in the mode of their approach—the hysteric patient gathers her robe around her, and falls gracefully; she appears to the casual observer to be unconscious, but there is not real or absolute loss of sense or of perception; there is not the hideous distortion of feature observed in epilepsy, nor is there the dilatation of the pupil; the eyelids may quiver, and the eyeballs may be turned up, but there is no divergent strabismus, nor is there the wide-open eye. Examined carefully, the physician may observe that the patient not only sees, but looks; the eyes are often definitely turned towards objects or persons standing near, and then rolled up again towards the forehead : there is no bitten tongue, although there may be much foaming and spluttering with the mouth: the breathing is tumultuous and noisy, but there is no such absolute arrest of respiration as to cause asphyxia; and the irregular movements and noises that accompany the laboured breathing may often be seen to be occasioned by the lips. The attacks last for an indefinite time, are followed by much apparent exhaustion, but not by real stupor.

Wherever the attacks pass beyond the description here given, it is probable that something more than mere Hysteria exists, and that the case borders upon the much more severe ailment known as epilepsy. In a few patients the two diseases co-exist, and then the attacks bear some of the characters common to the two elements; but in the vast majority of cases it is comparatively easy to distinguish between them.

Mr. R. B. Carter, in an interesting book,[1] has described the hysteric paroxysm under three phases, giving to them the terms "primary," "secondary," and "tertiary;" implying that in the first instance the attack is quite involuntary, and is the product of violent emotion; that in the second it is reproduced by association of ideas; and that in the third it is deliberately "got up" by the patient. There does not, however, appear to me to be sufficient reason for adopting these phrases, as I am sure that in many instances all that can be said of any of the tertiary paroxysms may be affirmed with equal accuracy of the very first attack.

Hysterical Mania sometimes appears after an attack, and its features resolve themselves into an exaggeration of the condition already described as the "hysteric state." The patient is unmanageable, sometimes mischievous, and very often highly abusive; but generally is merely loquacious, unreasonable, and demonstrative in regard of emotion, and the attack speedily subsides under judicious treatment. It, however, exhibits a great tendency to recur; and hysteric patients sometimes become, for a time, maniacal without going through a paroxysm of convulsion.

Hypochondriacal symptoms are met with, and are by no means rare, in cases of Hysteria; but it is quite easy to separate the two diseases.

PATHOLOGY.—Anatomical investigation has failed to show the presence of any organic lesion which is either so constant or so prevalent in Hysteria, that it may be justly regarded as its cause. Pathological examination has been equally unsuccessful in its attempts to explain the disease by a reference to the disturbed function of any one set of organs. It is common to find some derangement of the digestive, the assimilative, or of the reproductive systems; but these may exist without hysteria; and, *vice versâ*, that disease may be present when those bodily functions are healthily performed. There is, however, one thing common to all cases of Hysteria, and that is a perturbed condition of the nervous system. The essential character of this morbid state is an exaggeration of involuntary motility, and a diminution of the power of the will; the emotional, sensational, and reflex movements are in excess, while the voluntary are defective. The outcome of such a condition is seen in the mode of life of the hysteric patient. The will is determined by anything rather than by judgment, while ideas, feelings, and fancies exert an undue influence. Sensations are often morbidly acute, are uncorrected by any careful discrimination, and thus they increase the evil. Reflex movements, which in health are under some control, are not only exaggerated in their individual intensity, as a part of the hysteric state, but, from the weakness of volition, are allowed to run such riot that they pass beyond all bounds of healthy influence.

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[1] On the Pathology and Treatment of Hysteria, p. 43.

It has already been shown that the hysteric condition is somewhat analogous to that of the earliest period of decay, whether that be the result of age or of degeneration from disease. Hysterical symptoms are common enough in softening of the brain, and also during the course of, or convalescence from, exhausting diseases. They may break out suddenly, from a loss of blood, of food, or of rest, and they may occur as the immediate sequel of some violent shock, mental, moral, or corporeal. Whatever weakens the individual generally may bring about this state of nervous disturbance, may alter the relations of the several nervous functions; and this is, I believe, the true pathology of Hysteria, a disease which is more closely associated with affections of the nervous system than with those of the generative organs, although it is well known that the latter may and do exert a marked influence upon the former.

The hysteric state is essentially one of mental perturbation; and it is brought into existence, if not inherited, by those conditions which are the most active in producing disorder of the mind: in the male sex by worry, anxiety, over-work, late hours, accidental injuries, and dissipation; in the female sex by vexatious emotions, want of sympathy or success, disappointed and concealed affection, want of occupation, fear, and morbid conditions, or supposed morbid conditions, of the reproductive system. The latter are sometimes the coincidents, but I believe much more commonly the effects of Hysteria than its cause. Their relation is by no means constant in existence, and is most variable in kind.

It would appear that the nutrition of the whole nervous system is changed, but that the change is of such kind that it passes beyond our power of recognition, except in its physiological or pathological effects. We cannot see degeneration of tissue here, or too rapid a metamorphosis there; but we can witness the effects of such morbid processes, in movement, in secretion, and nutrition, and we can observe some of the ulterior results of such changes, in emotion and sensation.

The influences exerted by emotion upon secretion and nutrition have been well shown, in their relation to Hysteria, by Mr. Carter,[1] and the inter-relations of physical, mental, and moral life have been very ably treated by Mr. Hovell in a more recent publication;[2] but the primary fact in that condition which we term Hysteria, would seem to lie behind all that is referred to in these considerations, and to consist in that special morbid change of the nervous centres, which either gives to emotion an undue influence, or removes the limitations of its action. There are divers links in the chain of causes and effects, but there is one link at which, in fixing upon the pathology of Hysteria, we must stop and say—all behind this is cause, all beyond it is symptom or effect; here is the one point which determines the hysteric result. Up to this link we find the causes of chorea, of epilepsy, and of insanity, together with and identical with those of Hysteria; beyond it we find neither chorea, epilepsy, nor insanity, but what we term Hysteria: in that link, therefore, we must seek for and find, if we can, the essential fact of the disease. I do not say that we have found it; still we are much nearer its discovery than we were fifty years ago: but I think it better to state, in general terms, wherein this morbid condition lies, than to lose sight of that point, by regarding some outlying facts, and attaching undue importance to certain frequent lines of apparent causation and effect. There is nothing to prove that the vaso-motor or sympathetic system of nerve-fibres is primarily at fault; on the contrary, it often appears that the secretions and the general nutrition are late in suffering; and that the earliest departure from health is to be found in the disturbed balance of mental and emotional operations. If it be held that every change in every organ and every function is, more or less directly, determined by a change in the vaso-motor nerves, Hysteria may be driven theoretically into this "sympathetic" corner; but, when it is made to go there, it will find itself in company with almost every ll to which flesh is heir—with tubercle and corns, with cancer and ataxy. If the "vaso-motor system" furnishes the agency by which all departures from health find their expression, we have yet to discover the nature and cause of those special changes in its action which lead to these particular results.

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[1] Op. cit. p. 5 *et seq.*

[2] Medicine and Psychology, p. 56, &c. The gist of Mr. Hovell's argument lies in the following sentence:—"The nutrition of the body is not affected, mental power is not impaired, although it may be suspended, innervation is deranged, for the generation of nerve power is feeble, and its distribution is irregular; but it is the sympathetic, the vaso-motory system, the moral power, that is at fault: either from exhaustion of the physical strength of the sensori-motor centres, or because, perhaps most frequently, the purposes of life are in some respects disappointed, and the paresis of disappointment not

The most general expression that we can give to the pathology of the hysteric state is, perhaps, this, that it is a malnutrition of the nervous system, so distributed that its higher functions are relatively impaired and subordinated to the lower,—that there is diminished power of the former and increased activity of the latter.

Every one knows that, in health, there are numberless processes which are quite familiar to the mind, but which appear very strange when described in technical language. A physiological or pathological discovery is sometimes nothing more than the translation into scientific terms of a "well-known saw;" and what may now be said upon the pathology of Hysteria is little more than such translation. Slight emotional excitement, such as shyness, trifling vexation, or moderate pleasure, may flush the face, quiver the lip, and make the breathing "panting;" strong emotion, such as terror, intense anger,

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nly saps the strength, but, at the same time that it brings low the nervous system, lso renders it peculiarly liable to irritable excitability from opposing and aggravating uses."—P. 70, op. cit.

or disgust, may blanch the cheek, fix the jaw, parch the mouth, and hold the breath. Moderate distress may "find relief in tears;" but when grief is deepest the eyes are dry. The postures of the various emotions are known, and have been studies for the artist in marble or on canvas, and for the actor on the stage. If we express this physiologically, we say that emotions lead through certain nerve-tracks to the contraction or elongation of some muscular fibres: that the vessels are dilated in the one instance, so that more blood than usual passes through the organs; and are diminished in the other, so that the circulation is arrested; in the former, occasioning an excess of secretion, in the latter, a defect: that the muscles are spasmodically fixed in one condition, and in another are relaxed; but what we want to know is the primary fact leading to such changes, when they are unusual in degree or persistence, and morbid in kind.

Some individuals, we say, have more "control over themselves," or more "presence of mind," than others; that A never shows what he feels, while B never hides, and never can hide, anything; and if we translate this into technical phraseology, it is but to say that the one is of "phlegmatic," and the other of "nervous temperament;" that A is a stolid, resolute individual, and that B is somewhat "hysteric;" but here again we do but throw the question one step backward.

There is an old proverb to the effect that "it is the last straw that breaks the camel's back;" and if we put this into other terms, it often means but this—that an individual has for a long time gone on bearing what was too much to bear healthily, that he has struggled against it, and by forced effort has made everything appear quite tolerable or even easy; but at last he "breaks down" from some "shock," and then all the "wear and tear" comes out, and friends see that he had done or borne far too much before. Sometimes what is held to be "shock" is a mere trifle compared with the rough handling that had been previously endured without complaint; it is simply because it, a mere "straw," fell upon the already overburdened soul that the great crash came, and that then—all power of resistance being gone, the "back broken," as it were—the brave sufferer was prostrate, crushed, gave way, and the pent-up tide he had kept back so sternly broke through destructively.

In some there is weakness at the outset, congenital, or acquired; in others there is weakness, but it is induced by long patience, vexation, care, or trouble, that have at last done their work, and a work which it may take years to undo. But here, again, we have yet to learn why, out of a hundred individuals similarly exposed, one becomes hysteric, another epileptic, and a third maniacal.

If we refer to the etiology of Hysteria, we shall see that all the facts point in one direction; if we look to the symptoms, we shall see that they are partially explained. In the female sex, at certain ages and under certain conditions, Hysteria is most common, for it is but an exaggeration of that which constitutes the normal characteristics of that sex; in the male sex it is met with when circumstances have gradually converted males into the condition of the other sex; *i.e.* when emotions have been so played upon that they have, at last, broken through the force of resistance, which held out for a long time bravely, but at length gave way.

The essential fact of Hysteria, then, is the distorted balance between voluntary and involuntary power; volition is defective; emotional, sensational, and reflex activity are in excess; and this distortion may be brought about by the many and divers circumstances of age, sex, position, employment, and the like which have been enumerated in the section on etiology; but the precise nature of the change which is the efficient cause of such distortion—*i. e.* the primary physical fact in the pathology of Hysteria—has yet to be discovered.

DIAGNOSIS.—If the symptoms already described be borne in mind, and the history of each case be carefully considered, there is not much difficulty in the diagnosis. A physician called in on an emergency may have, however, to distinguish Hysteria from several diseases which it simulates.

From *Epilepsy* it may be separated by negative characters. There is neither the suddenness of attack, the absolute loss of consciousness, the dilated pupils, the complete asphyxia, the bitten tongue, nor the reckless injury of either the person or the clothes. The patient "looks about," the attack lasts longer, there is much sobbing and crying, much exhaustion, but no perfect stupor. The interparoxysmal state of the hysteric patient exhibits features not met with in epilepsy, and *vice versâ.*

From various inflammatory affections, such as *Peritonitis, Laryngitis,* and *Arthritis,* hysterical symptoms may be distinguished by a careful use of the thermometer, which fails to show any rise of temperature. Again, the tenderness of parts complained of may be seen to be ideal rather than real, and to bear relation to the skin quite as much as, and often more obviously than, to the deeper tissues. When there is loss of voice, this has the characters already described; there is no attempt made to whisper loudly; the failure is evidently not in the apparatus of voice, as a mechanical production, but in the will to put that machinery into play. A laryngoscopic examination will show that the throat is healthy, or is simply relaxed—the vocal cords being widely separated, and slight effort being made for their approximation. The pulse-respiration ratio will further show that although the breathing may be tumultuous, there is no real dyspnœa. *Phantom tumours* may be removed by the inhalation of chloroform, while palpation and percussion usually reveal the nature of their constituents.

Organic diseases of the nervous centres, which are sometimes simulated by hysteric paralyses and anæsthesiæ, may be excluded by the conditions already described, when detailing, in the section on symptoms, the mode of their development. Usually the phenomena presented are inconsistent with the idea of any definite disease of either the cerebrum or the spine; the paralyses are imperfect in development, vague in their distribution, and changing in their locality; they are not accompanied by the alterations of nutrition, or of electric contractility or sensibility which are proper to other affections; and the history of the case will usually reveal their true nature. The walk in hysteric paralysis has already been described; but it is further to be observed that the patient does not look at her feet, as those who are ataxic do, but looks round about her to observe the effect of her performance. The ataxic or paraplegic patient tries to walk; the hysteric girl tries to show that she cannot use her limbs: if the former forgets himself, he falls; if the latter forgets herself, she walks.

Neuralgia, when of hysteric origin, has not the real intensity of the genuine disease, as may be proved by withdrawal of attention. There is also an absence of those "painful spots" which are present when Hysteria does not complicate the case. The distribution of pain described by hysteric patients is, moreover, often so wide of all relation to anatomy and physiology, that its true nature may be recognised.

PROGNOSIS.—When once established, Hysteria is very difficult to cure, and this is true under all the conditions of causation. The most difficult cases are those in which it is but an exaggeration of a constitutional defect, inasmuch as it is impossible to cure the malady without changing the individual, and this is by no means an easy task. If the hysteric habit be natural, or have become a "second nature" by long existence, the prognosis is *pro tanto* bad: if it be something quite unlike "the former self," or if it have been only recently developed, there is much room for hope. If the cause exist in the patient, *i.e.* in the essential features of the individual character, comparatively little can be done; if it lie in external circumstances, much may be done, provided that those circumstances can be changed. If there be definite organic disease, and this be of such a character that it is amenable to treatment, prognosis is so far favourable; but if there be no such disease, and *à fortiori* if the general health be good, the prognosis is unfavourable.

Hysteric symptoms, such as paralysis, aphonia, and the like, are often easily removed when they are of recent origin; but when they have existed for many months, the prognosis with regard to them is unfavourable. It is not, however, hopeless; for in some cases, of even many years' duration, there has been amendment, and—but more rarely—cure.

The prognosis in Hysteria depends, therefore, mainly upon these two things—the nature of the "cause," and the freedom with which treatment may be employed. No human being can cure, the physician can do but little for, one who is born hysteric; *i.e.* for one whose disease is but an exaggeration, and sometimes only a slight exaggeration, of her habitual, constitutional state. Education might have accomplished much in childhood, but often when the physician i called in, the grooves of life are worn so deeply that he cannot chang them, and all that is possible is to soften their sharp edges, or to retard the movement which he can neither stop nor guide. In such cases the prognosis is unfavourable. When Hysteria is an accident, is unlike the earlier promise of the individual, or when it is brought about by long, and at last unendurable pressure from without, then there is much room for hope. When the treatment of the hysteric patient is cramped by the anxieties of friends—limited, in this direction and in that, by frightened, too sympathetic, or unwise relatives —the prognosis is unfavourable; but when the circumstances are such that the physician can control them all, much hope may be entertained. Upon these points, rather than upon the special character of the symptoms, the prognosis turns. When Hysteria is a disease, and the physician has given to him a *carte blanche* to treat it as he deems best, the patient may be cured: but when it is a constitutional peculiarity, and the physician is checked at every turn by anxious friends, the case is hopeless, and might as well be left alone.

In regard to all special symptoms, the prognosis is more favourable than it is in those diseases which Hysteria simulates; but even here the general principles just stated are the most trustworthy guides in our attempts to forecast the future.

TREATMENT.—The old copy-book maxim, "Prevention is better than cure," expresses but a small portion of the truth in regard to the management of Hysteria: prevention might be easy; cure is often almost impossible. It is not within the scope of this work to describe generally the processes of a healthy education, but some things that are special must be said with regard to prophylaxis.

Bearing in mind the pathology of the disease, it is highly important that its earliest indications should be recognised and combated. Where there is the tendency to Hysteria in early life, these things are necessary:—

1st. A strenuous effort to draw the person "out of himself," or "herself," and to develop the faculty of self-control. This should be done, not as an occasional or spasmodic effort, but as the business and prevailing arrangement of daily life; and while it is done, and in order that it may be done, the predisposed person should be unconscious of the process. A child who is "peculiar," disposed to be taciturn, loquacious, "excited," or unduly gay; or who is very readily "upset," and is "so sensitive" that parents and others are "afraid to tell her" this or that, "for fear that it should make her ill;"—one who is "impulsive," and disposed to find fault with herself without just ground; one who is "shy," and hides herself; one who is morose, and who thinks herself "misunderstood;" or one who is retiring, and shuts herself out from the sympathy she craves for;—should be carefully watched, tended, and unconsciously guided away from self, and into some line of feeling, thought, and action, which may interest the mind without fatiguing the body. The worst thing that can be done is that which makes the patient know and feel that she is thought to be "peculiar." Sometimes such treatment is gratifying to her, and she likes it—it is easy, and it "seems kind" to give it—but it is radically wrong. Anything that looks like harshness, rigid discipline with a view to improvement, or want of sympathy from want of "understanding her feelings," is shrunk from, or resented by, the patient, and is worse than useless. At the same time, all exhibitions of a want of self-control should be checked, and much of this kind may be done in the nursery, and long before Hysteria is dreamt of. Kindness, firmness, and obvious recognition of "tender points," with judicious, sympathetic, and wise regard to them, may do much to avoid future evil; and sometimes the qualities of mind which will produce such treatment may be met with in a parent, a sister, or a governess. The physician should guide, in little details of daily life, those who have the management of such a child. The object is to make her feel that she is understood and cared for; and the best mode of attaining this object is often indirect in its operation. The mind and the heart should be engaged in some healthy pursuit; interest should be awakened, and exercised in anything—it matters not what—outside the individual; self should be lost sight of, and life made useful.

2nd. The bodily health should be most carefully regarded, and this without any admission or appearance of anxiety on the part of others. The points that require attention are the following:—(*a*) *Diet*, which should be sufficient, wholesome, and easy of digestion, avoiding too long an interval between meals, and observing an especial regularity in the times at which they are taken. (*b*) *Rest*. The hours of sleep should be long, and those of rest or lying down not too long. Hysteric girls, or those who are disposed to become such, are in the habit of reading at night and of lying in bed in the morning. Such habits should be broken,—not roughly, for the sake of breaking them, and of "doing something disagreeable," but—by supplying a reason and a motive for a different mode of life. (*c*) *Exercise* should be taken regularly, and in the open air, as much as possible without fatigue; and here, again, the exercise should have some other apparent object than a mere piece of tedious hygiene. (*d*) *Recreation* should be ample and merry; but all such things as precocious or preternatural excitements should be avoided. (*e*) *Study* should be systematic and disciplinary, but varied and interesting, and made to subserve some purpose which lies, obviously, outside of mere personal accomplishment or pleasure. (*f*) The various functions of secretion, excretion, and (if they have commenced) of menstruation, should be regulated; and this, again, should be done, as far as possible, without any particular notice being taken of the fact that they may be unhealthy.

3rd. Some motive or purpose should be supplied which may give force, persistence, unity, and success to the endeavours of the patient. This is sometimes very difficult to manage; but it is less difficult in early life and in predisposed persons than it is in those who are older and have already shown definite symptoms of the malady. Still, much may be done by those who have a little ingenuity in detecting character, and a great deal of perseverance and determination in carrying out their wishes. The patient should be led to feel that the object is in itself desirable; she should never think that it is suggested for her own treatment or benefit. If she is urged on the former ground, she may do much; if on the latter, she fails entirely. The hysteric patient is often most thankful for and happy in the idea that she is doing good to others, and she will take great pains to make her efforts successful and pleasing; but she hates the notion of doing anything of such kind as a mere means of self-cure, thinks that the doctor who recommends them is grossly ignorant of her real wants, and that the friends who urge her onwards are singularly stupid or unkind in their advice. To "make an effort," simply because told to do so for her own sake, is sheerly detestable to the hysteric patient, and is sometimes as impossible as it is distasteful; but to exert herself, almost unconsciously, because a motive is supplied, is scarcely felt to be an effort. The patient does what she herself is surprised and pleased with, and derives great benefit from the process.

When the symptoms of Hysteria are developed, the treatment should be conducted upon the principles already laid down with regard to prevention. There is, so far as I know, not one single drug which exerts any specific action on the disease; but there are many medicines which may be used with advantage for the relief of associated disorders. These do not require any special notice here, further than to say that if the patient be anæmic, iron is useful; if deficient in general nervous tone, quinine, strychnia, and vegetable bitters may do good; if there be indigestion with much flatulence, bismuth, charcoal, and alkalies, or mineral acids with light bitter infusions, may give relief; if there be constipation, mild aperients may overcome the difficulty; if there be menstrual derangements, they should be treated upon general principles.

The whole list of anti-hysteric remedies—such as musk, castor, valerian, asafœtida, and the like—appear to have this one property in common, that they do no good, and delay the real treatment of the case, which is not one of "nauseous gums," but of mental, moral, and social management.

Painstaking appreciation of the patient's own feelings; determinate assurance that the disease is a real thing, and no idle fancy; strenuous effort to help the patient in weakness, and to set her right when wrong; fertility of resource in little things; a cheerful but not boastful, a sympathetic and calm, but neither condoling nor anxious, manner; and a strong will, with patient work and tact, may do very much, and may often cure. But the physician should see that he manages his patient, and that all that he has attempted to effect is not undone by frightened relatives or anxious friends.

There are some drugs which are useful under special circumstances; and these are opium, or morphia, where there is loss of sleep, or persistent pain; and the diffusible stimulants, where there is a great tendency to recurrent spasm. Morphia may be administered most effectually, for the relief of pain, by hypodermic injection: it may be given by the mouth, with light food, when there is want of sleep. Chloric ether, ammonia, and musk, often relieve the tendency to spasm; and in some cases Indian hemp has proved of service when other medicines have failed: in the large majority of cases, however, I have found Indian hemp of but little use. Asafœtida, in doses of thirty grains, three or four times daily, is of service in some cases. Bromide of potassium has appeared to me to be singularly useless in Hysteria, failing to relieve either the attacks or the symptoms which exist between the periods of their recurrence.

The *attacks* of hysteric convulsion may be arrested by a plan suggested by Dr. Hare—viz. that of forcibly preventing the patient from breathing for a certain time, by holding the nose and mouth. The effect of such constraint is to make the patient, when allowed to do so, "draw a long breath," this vigorous inspiration being usually followed by a relaxation of all spasm, and a disappearance of the fit. Some attacks are of such short duration, that there is neither occasion nor time for this mode of treatment; but when they are prolonged, I have seen it notably useful.

Dashing cold water on the face and neck may sometimes succeed in doing imperfectly that which Dr. Hare's treatment accomplishes effectually; but even cold water is not always at hand, and when it is—in addition to its other inconveniences in regard to carpets and dress—it often fails to do any good.

A calm manner, the absence of all appearance of alarm, and of either scolding or distressing sympathy,—all of which things the apparently unconscious patient observes much more accurately than do her frightened friends,—will sometimes bring a fit to a speedy end.

Some *special symptoms* of Hysteria require special treatment. *Aphonia* may often be cured by electricity; and the mode of application which has appeared to be the most useful is that of giving sparks to, or taking them from, the larnyx. An ordinary plate or cylinder machine may be used for the purpose, and either the patient or the physician may be insulated, and the sparks taken from or given respectively by a brass knob. The interrupted current from a magneto-electric or volta-dynamic apparatus may be used so as to pass the shocks through the throat, or a shock may be administered from a charged Leyden phial. Under all these circumstances, the voice is sometimes instantaneously restored. But when Aphonia has been of very long duration, and such measures fail to affect it, good may be done by directly galvanizing the vocal cords, in the manner proposed by Dr. Morell Mackenzie. Further, I have known a strip of blister round the throat recall the voice when all means of electrifying have failed.

Paralyses are treated very successfully by Faradisation, and by passive movements and frictions, employed by a well-instructed nurse.

The electricity should be applied to the muscles affected, and also to the skin which covers them. I have, however, found no mode of treating hysterical paralyses comparable in efficiency with that of placing narrow strips of blister completely round the affected limbs. This method of treatment has succeeded perfectly and rapidly, after all other plans have failed.

Rigid Contractions may be relieved by the continuous galvanic current, but much more successfully by the inhalation of chloroform, and the adaptation of some apparatus to maintain extension when the effect of chloroform has subsided. Passive movements are also of much service in such cases. Drugs may be taken in almost poisonous doses without relieving tonic spasm.

The treatment of other symptoms must be conducted upon the general principles already laid down, and may be assisted by those local sedative measures to which allusion has been made.

ECSTASY.

By Thomas King Chambers, M.D. F.R.C.P.

By intense concentration on one object, engaging only a few of the intellectual faculties, the mind is liable to lose temporarily its sensitive and controlling power in respect to its other relations. In minor degrees this state is a matter of daily experience with us all, and in minds of average strength it does not seem to go beyond minor degrees. They do not wish or practise such intense concentration; they are able to do and feel all they want to do and feel without overtasking themselves. But there are some, either naturally weaker, and so incapable of full feeling without concentration, or else desirous of a higher degree of emotion than they are healthily capable of; and in these a condition may be adduced allied in some respects to catalepsy, and in some to hysteria—a condition certainly morbid, for it renders the patient unequal to the functions of social life, and is excited by causes which affect some and not others.

Sometimes the patient falls into a state of immobility, in which there is a passive reception of ideas, like that of the Midianite prophet "falling into a trance, but having his eyes open," a state he evidently considered rarer than that of a mere dreamer of dreams. Hoffman[1] describes an ignorant peasant woman of twenty-four, after a fortnight's course of exciting sermons, remaining motionless for more than an hour; after which she gave a few sighs and returned to herself, having seen or heard nothing of what was going on about her, but having had exquisite revelations of the love of Christ. During forty days she had a hundred returns of the same state, which would always be induced by a recitation of a few verses of the Scriptures concerning the love of God. During the fits the pulse remained quite natural. They were finally removed by a change of air and scene, after bleeding and stimulants had failed. The followers of St. Francis, in the days of faith, were often brought into these ecstasies by fasting, meditation, and abstinence from varied intellectual exertion. Nowhere is it so strikingly depicted as by the Tuscan artist Cigoli—a man of by no means lively fancy, for, except an Ecce Homo, he painted nothing well but ecstatic and starving Franciscans. So he is probably truthful. After visiting each Florentine gallery it is difficult to expel from one's memory these strange figures, of marble paleness, kneeling, but sunk on one side from exhaustion, the eyes open, the pupils fixed, the arms extended to embrace the beloved vision, the livid lips parted in smiles, showing the parched dark mouth, the breast heaving with delight. It is necessary to add only one medical fact, derived from M. Sagar's observation[1] of a Capuchin in this state; namely, that the pulse was pretty strong.

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[1] Medicina Rationalis, vol. iii. p. 50.

One main psychical difference between this and catalepsy lies in the visions which are recollected afterwards with all the force of reality. In true catalepsy all memory of what is done during the fits is completely wiped away—a phenomenon which may assist in the detection of impostors.

A more common development of Ecstasy is where the sufferer feels "borne in upon him," a desire to communicate to others the feelings he is sensible of himself, instead of reserving the experience till afterwards. The simplest instances of this are the scenes which take place from time to time in the Primitive Methodists' or "Ranters'" chapels. The congregation groan and respond to the feelings of the preacher, second his words with their own experience, and various members work themselves up into a state of excitement, repeating the last words of the sentence, "Salvation! salvation!" or whatever else it may be, with continually increasing earnestness, till they end in shouting and sobbing.

The next stage or form of Ecstasy is where the enthusiasm tries to express and exhaust itself in bodily movements. One of the most famous instances of this is the spasm of the *Convulsionnaires de Saint-Medard*, a disease which was by neglect allowed to attain most formidable proportions in the last century. It owed its origin to the discussion of dogmas whose character one would never have expected to have stimulated feeling; namely, those which were in dispute between the Gallicans and Ultramontanists on the occasion of the bull "Unigenitus." A popular Gallican deacon, named Pâris, died in 1727, and was buried at St. Medard; and on his grave people began to fall into convulsions, be affected with *clairvoyance*, preach, jump, spin round with incredible rapidity, run their heads against walls, &c. &c. Of no avail was Louis the Fifteenth's proclamation, which some one parodied—

> "De part le Roi! Défense à Dieu
> De faire miracles dans ce lieu."

In spite of it Convulsionism grew into a sect, and was at last only smothered by the French Revolution.

A transition between this form and the last is exhibited in Revivalism; and its reduction to a certain system and order is shown in the ceremonies of the Jumpers of New York and the Dancing Dervishes of Cairo.

Sporadic cases of this Ecstasy not infrequently occur in the experience of the promulgators of new or arousing doctrine; but judicious discouragement has usually prevented its becoming historical. We meet with it in the biographies of such men as Samuel Wesley and Edward Irving; and a valuable lesson is to be learnt from their wise mode of dealing with it, especially the former.

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[1] Quoted by M. Tissot, Œuvres, vol. xiii. p. 4.

The mention made of this disease being used by dishonest or foolish people as a bond of union for religious sects, leads to a point in its history which constitutes the main interest it possesses for practical consideration. It is eminently communicable, especially in its more active, noisy, and ridiculous forms.

There is not much worth reading in the Pastorals of Longus; but one expression of his—ἡ τῶν ὀφθαλμῶν ἅλωσις "the contagion of the eyes"—is so picturesque and truthful that he deserves the credit of it. Through the eye instinctive imitation, or sympathy, directs the first intellectual and corporeal efforts of the infant, and makes him grow up in the image of his kind; and, as Longus felt, it is by such means that two adult souls get bound into one. So also through the eye flashes in that morbid state in which a nervous malady fetters the normal powers of control, binding them up as with an electric spasm, and allowing the lowest animal emotions to exhibit themselves. An initiative compliance, a voluntary surrender of the gates of the soul, is doubtless necessary at first; but with each yielding the energy is weaker, and this natural and healthy sympathy may pass into an actual disease of the mind, in which the power of the will is quite in abeyance.

In this way, from the accidental eccentricity, convulsion, or insanity of often a single person, the strange spasmodic epidemics of the Middle Ages arose. We can easily understand the disorganization which they produced among the lower orders, when we read that a few months after a new appearance of the "dancing mania" at Aix-la-Chapelle, on St. John's Day, 1374, there were as many as eleven thousand dancers in the streets of Metz. In this instance, at least, the outbreak took its rise in the scandalous midsummer revels, which had been handed over from paganism to the ascetic Baptist's festival, in spite of the protests of the Church from St. Augustine to Pope Boniface. The origin was a disreputable one, so the dancers hastened to avoid the inference by placing themselves under the protection of St. Vitus, one of the fourteen "Helpers in need."[1] For full 150 years it was in Germany a most serious plague, of which a full history is given in Hecker's "Epidemics of the Middle Ages." As a translation of that graphic description is published by the Sydenham Society, it is unnecessary to do more than refer to it those who are anxious to trace to its most disgusting results an extreme indulgence in uncontrolled sympathy.

Spasmodic epidemics seem to have appeared in Italy about the same time as in Germany, but to have been for some time confined to Apulia. At the end of the fifteenth century they spread further, and coincident with the spread there seems to have been observed an increase in the numbers of the tarantula or ground-spider. The two facts became associated together, and a panic flashed like lightning through the country that this creature was communicating the disease by its bite. Of "tarantism" in Italy, pure fright was as potent an exciting cause as superstitious fanaticism had been of the "St. John's" or "St. Vitus' dance" in Germany. And for this reason it affected a higher and better educated class of society. Even a sceptical prelate did not find his freethinking a protection. Quinzato, Bishop of Foligno, having allowed himself in joke to be bitten by a tarantula, fell into the disease, and could only be cured in the undignified method adopted by vulgar laymen. (Hecker.) He was obliged to kick off his shoes (such at least is the necessary prelude nowadays) and dance the tarantella.

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[1] Most travellers in Flanders and Germany are probably familiar with the shrines of the fourteen "Nothhelfer" or "Apotheker" saints, spiritual specialists, of whom one cures toothache, another stone, another cancer. It is a curious fact that the regularly educated physicians, St. Luke, St. Cosmo, and St. Damian, are not among them.

As an epidemic, tarantism has long disappeared, but sporadic cases are said still to occur, and hysterical women will persuade their gossips that they have been bitten by the tarantula, and that they cannot get rid of their mental fidgets without an immoderate indulgence in the traditional cure by dancing.

Of the exciting causes of Ecstasy, and its allied spasmodic epidemics, the most common is perverted religious feeling, of which elaborate examples are given in the "New America" and other works of Mr. Hepworth Dixon. The reason is that, since the diffusion of Christianity, religion has a more powerful hold upon the emotions than anything else. But all historians agree in attributing much influence also to venereal excitement; and in pre-Christian times, when in default of revelation men worshipped their incarnate passions, we have from the pen of Sappho a description of a purely erotic ecstasy, which can never be paralleled again. In the case of tarantism it seems to have been cowardice which was the exciting cause.

The history of the treatment of exaggerated instances of this disease is instructive to us, not from the likelihood that any of this volume's readers will be called upon to undertake it, but as a suggestive guide for the management of allied states, semi-mental, semi-corporeal, which are as commonly, as it is rarely, under medical charge. Allusion is especially intended to two—*chorea*, the heir to the name, and in a diluted degree to the nature, of the mediæval mad dancers who put themselves under the protection of St. Vitus; and to *hysteria*, which, being more usual among women than among men, has acquired a name derived from a part of their bodies anciently supposed to produce the symptoms—a nomenclature often leading to bad practice.

1. If taken in time, ecstatic and emotional exhibitions are capable of being forcibly repressed. For example, in Unst, the most northerly of the Shetlands, an epidemic of convulsive fits occurring in sermon time began to prevail in several parish churches. At one of these, Northmaven, the disease was cut short by a rough fellow of a kirk

officer, who carried out a troublesome patient and "tossed her into a wet ditch." Nobody else caught it.[1] From what scandalous scenes Europe might have been saved had the first dancers on St. John's eve been "tossed into a wet ditch!"

2. A strict quarantine prevents infection. In 1796 an epidemic convulsion spread to twenty-four persons in Anglesea. Their landlord, Lord Uxbridge, consulted Dr. Haygarth, and by his advice all communication with the afflicted persons was prevented, and the plague was stayed, as he records.[2]

I have often had chorea and hysteria arrested in hospital wards on separating the patients thus diseased, who had been keeping up one another's malady by sympathy.

3. Order, rhythm, designed consecutiveness, and in short anything implying voluntary control, has a beneficial effect in this class of complaints. Doubtless when once Sappho had grown particular in winding up with a dactyle and spondee the pretty stanzas named after her, when Madame Guyon had learnt to be careful of her rhymes, and Saint Theresa had committed her devotions to paper, all danger of contagious enthusiasm was past. But it is only an intellect of above the average capacity that can undertake to reduce itself to order in this way. Those afflicted in Germany with the epidemic convulsions before alluded to, took to dancing, evidently with an instinctive feeling that rhythmical movement was a relief to their morbid sensations; but in that country the application of it as a mode of cure does not appear to have been turned to such good account by art as in Italy. The tarantellas I have seen performed in South Italy are very complicated figures, accompanied by an amount of arm-waving, finger-snapping, simultaneous wriggling, slapping of hands, bumping of backs, and crossing one another's footsteps, that can only be accomplished by a strict adherence to time. And the time is marked by a tambourine or drum. These dances are probably much older than tarantism; but for the cure of it they became popular, and from it they got their name. It is impossible to doubt that they had a real influence, even over those who undertook them unwillingly, as in the case of the sceptical bishop above alluded to. The peculiar features of them, to which I should attribute their usefulness, are the marked time and intricate figure, by which they are honourably distinguished from the senseless rotatory embrace now called dancing.

I am sure I have seen decided benefit in hysteria from dancing reels, and there would probably be much more, if time were better marked and kept.

In chorea, marching in timed step is excellent practice for regaining the directing power over the limbs. In stuttering, which is a sort of chorea, spouting poetry before a looking-glass contributes much to the cure.

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[1] Edinburgh Med. and Surg. Journal, vol. iii. p. 439.

[2] Haygarth, "On the Imagination as a Cause and Cure of the Disorders of the Body." Bath, 1801.

CATALEPSY.

By Thomas King Chambers, M.D., F.R.C.P.

Name.—This word Catalepsy is derived from the Greek κατάληψις a "seizure" or "arrest."

Definition.—Catalepsy is the name given to intermittent attacks of a suspension, more or less complete, of sensation and voluntary power, without convulsions, accompanied by a stiffening, general or partial, of the muscular system; so that the parts affected retain for a period of variable duration the position in which they happen to be at the invasion of the fit.

Description.—This is one of those pathological phenomena of whose anatomical cause we are entirely ignorant, and therefore it is best defined by its symptoms, instead of committing ourselves to any vague theory of its nature. It is best to call "Catalepsy" any attack which fulfils the conditions above named, and then we shall at once avoid all those discussions with which writings on the subject are laden about "true" Catalepsy and "false" Catalepsy, and the separation into symptomatic and idiopathic. which we have no warrant for making at all. It is as much Catalepsy, and the ultimate morbid state is essentially the same, whether it is followed by a disease with another name, or whether it is followed by restored health.

The following account of a well-marked case by Dr. John Jebb describes the details of Catalepsy more graphically and fully than any I have yet read. He says:[1]—

"In the latter end of last year (viz. 1781), I was desired to visit a young lady who for nine months had been afflicted with that singular disorder termed a Catalepsy. Although she was prepared for my visit, she was seized with the disorder as soon as my arrival was announced. She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form a figure of death-like sleep, beyond the power of art to imitate, or the imagination to conceive. Her forehead was serene, her features perfectly composed. The paleness of her colour, her breathing at a distance being also scarce perceptible, operated in rendering the similitude to marble more exact and striking. The positions of her fingers, hands, and arms, were altered with difficulty, but they preserved every form of flexure they acquired; nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it as firmly as her limbs.

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[1] Appendix to Select Cases of the Disorder commonly called the Paralysis of the Lower Extremities, by John Jebb, M.D. F.R.S. London, 1782.

"Upon gently raising the eyelids, they immediately closed, with a degree of spasm. The iris contracted upon the approach of a candle, as if in a state of vigilance; the eyeball itself was slightly agitated with a tremulous motion, not discernible when the eyelid had descended.

"About half an hour after my arrival, the rigidity in her limbs and statue-like appearance being yet unaltered, she sang three plaintive songs, in a tone of voice so elegantly expressive, and with such affecting modulation, as evidently pointed out how much the most powerful passion of the mind was concerned in the production of her disorder, as indeed her history confirmed. In a few minutes afterwards she sighed deeply, and the spasm in her limbs was immediately relaxed. She complained that she could not open her eyes, her hands grew cold, a general tremor followed; but, in a few seconds, recovering entirely her recollection and powers of motion, she entered into a detail of her symptoms and a history of her complaints.

"She informed me that she had no recollection whatever of what passed in the fits; that upon coming out of them she felt fatigued, in proportion to the time of their continuance; and that they sometimes lasted for five hours, though generally for a much shorter period.

"She further related, that the fits returned once or twice a day, sometimes more frequently; but that she was never troubled with them in the night. She sometimes lost her sight and speech, the power over her limbs and her intellectual faculties remaining unimpaired. The fits frequently attacked her without any previous warning; at other times, a fluttering at her stomach, and a fixed pain at the top of her head, occupying a part she could cover with her finger, announced their approach.

"Hysterical risings in her throat, appearances of fire, pains in her eyes, and not infrequently in her teeth, flatulence, a sense of weight in her stomach after eating, with convulsive motions in the region of that organ, were superadded symptoms of which she much complained.

"Her disorder was evidently exasperated at the approach of the catamenia, which were constantly present at the regular period. She was always much agitated previously to a storm of thunder, and every material alteration of the weather produced a sensible effect.

"After she had discoursed for some time with apparent calmness, the universal spasm suddenly returned. Her features now assumed a different form, denoting a mind strongly impressed with anxiety and apprehension. At times she uttered short and vehement exclamations, in a piercing tone of voice, expressive of the passions that agitated her mind, her hands being strongly locked in each other, and all her muscles, those subservient to speech excepted, being affected with the same rigidity as before.

"During the time of my attendance similar appearances were frequently exhibited.

"I was informed by the family of many particularities in the access of the disorder, all denoting its instantaneous effect upon the nervous system. She once was seized in my presence while drinking tea, and became universally rigid at the instant she was advancing the tea-cup to her mouth. Her tears sometimes flowed copiously, while every internal, as well as external, sense seemed entirely locked up in sleep.

"I will now proceed to describe the progress of the disorder, and the mode of treatment, before she was entrusted to my care.

"It appears that for many years before the access of the cataleptical symptoms, she had suffered much from violent headaches, particularly that species of headache termed clavus hystericus. Her spirits were easily discomposed. Her fingers, upon touching cold substances, would frequently lose their natural heat and feeling. Her habit of body had been uncommonly costive, but of late her bowels were much disturbed by every kind of laxative. Her nervous complaints were always particularly troublesome at the approach of rain and after a sleepless night.

"Her disorder commenced with hysteric fits; to these succeeded a delirium of several days' continuance, attended with slight shiverings, but no other sign of fever; the Catalepsy followed next in order, which at first affected her with only single fits, at a week or fortnight's interval; these gradually advanced in strength and frequency until by her own sufferings, and her sensibility on account of the anxiety of her friends, she was reduced to the most pitiable distress."

Then follow details of the treatment advised by Dr. Jebb, which was judicious and successful. An opium plaster to the epigastrium did good, but the last and longest continued prescription consisted of bark, gentian, and tincture of lavender, which she went on with till quite well.

This exceedingly well-drawn-up description makes one regret that the accomplished author had not continued to apply his pen to depicting the eternal truths of nature, instead of wasting it upon theological and political advocacy. It renders needless the repetition of stock cases which usually illustrate the subject.

Causes.—The most common exciting cause of Catalepsy seems to be strong mental emotion. When Covent Garden Theatre was last burnt down, the blaze flashed in at the uncurtained windows of St. Mary's Hospital. One of my patients, a girl of twenty, recovering from low fever, was woke up by it, and exclaimed that the day of judgment was come. She remained in an excited state all night, and the next morning grew gradually stiff, like a corpse, whispering before she became quite insensible that she was dead. If her arm was raised, it remained extended in the position in which it was placed for several minutes, and then slowly subsided. The inelastic kind of way in which it retained its position for a time, and then gradually yielded to the force of gravity, reminded one more of a wax figure than of the marble, to which Dr. Jebb compares it. A strange effect was produced by opening the eyelid of one eye; the other eye remained closed, and the raised lid after a time fell very slowly, like the arm. A better superficial representation of death it is difficult to conceive. The pupils, however, contracted sluggishly under the influence of light; and the pulse could be felt beating softly at both heart and wrist. She came round again by degrees in the course of the morning, and had no relapse; nor had she any manifestations of ordinary hysteria during her stay in the hospital, I believe.

Less acute, but more long-continued mental emotion will sometimes cause it. The same year as that in which the last case occurred, I was attending for menorrhagia from relaxed fibre a young woman, aged twenty-two or twenty-three, who had been a governess in a family I was acquainted with. She was of an affectionate disposition, and had been rather coldly treated,—"misunderstood," as it is called. The menorrhagia, too, had pulled her down a good deal, and forced her into involuntary idleness. One morning when I called to see her, she was in bed, unable to move, and scarcely capable of articulating. She said she was just recovering consciousness, but all the limbs were partially stiff, and the neck and back quite so, as appeared by raising her up with the hand at the back of the neck, when the body remained straight, resting on the heels. This state soon passed off, even while I was in the room. But the next morning I found her partially affected in the same way; the left side was rigid, and especially the left arm, which remained stretched out at an angle when so placed.[1] I observed that when I bent the arm, the deltoid contracted as it does when flexion is made by voluntary effort. She then told me that, though quite incapable of moving the limb of her own unassisted will, she thought she could do so if I bade her very strongly. And such proved to be the fact; for, on my rating her soundly and ordering her to get up, she at last obeyed. I explained to her what I believed to be the nature of the disorder, namely, a broken connexion (to speak metaphorically) between the will and the nervous system; and that she must rejoin this broken link by painful exertion and violent determination. She had no further relapse.

In both these cases I convinced myself carefully that there was no deception.

Catalepsy is sometimes very brief and sudden. I have a young lady now under my care, for non-assimilative indigestion, of whom I received the following accounts from a mother of more than ordinary intelligence and powers of observation. She said that her daughter was fond of reading aloud, and that sometimes in the middle of a sentence the voice would suddenly stop, a peculiar stiffness of the whole body would come on and fix the limbs immovably for several minutes. Then it would relax, and the reading would be continued at the very word it stopped at, the patient being quite unconscious that a parenthesis had been snipped out of her existence, or that aught strange had happened. She grew much better under tonic and restorative treatment, and gradually ceased to have these singular attacks; but after about a month's interval, as she was one evening engaged in playing a round game of cards, she suddenly went off into a regular epileptic fit, which was followed by sleep, and she did not recover consciousness till the next morning. This fit could be accounted for by certain errors in digestion, and she has had no recurrence of it, or of the Catalepsy, though four months have passed over. So I hope it was epilepsy of an intercurrent or curable sort.

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[1] M. Tissot mentions a similar case of partial Catalepsy affecting the arms in a man. (Œuvres, tom. xiii. p. 56.)

But sometimes the epilepsy preceded by Catalepsy is of a more serious sort. I remember a much-respected lecturer in this metropolis, in whom the *petit mal* of epilepsy assumed this form. He used to be attacked sometimes in the middle of a sentence, with his hand wielded in demonstration before his class. He would remain perfectly stiff for a minute or so, with mouth open and arm extended, and then resume his sentence just where he had dropped it, quite unconscious that anything had happened. After a time the seizures assumed the more usual and more fatal form.

This sort of short attack is not, however, always the precursor of anything so serious. Nor, if traceable to a material cause, is that cause necessarily in the brain. Van Swieten tells a story of a woman, forty years of age, who was roasting chestnuts in the frying-pan, and kept continually stirring them lest they should be too much scorched; in doing which, she was seized with a true Catalepsy. As Van Swieten lived hard by, he was immediately called in; in his presence she suddenly vomited two live worms, and forthwith proceeded with her cookery, quite unconscious of what had happened. She had no relapse.[1]

Other cases are of much longer duration. The death-like state may last for days. It may be mistaken for real death, and treated as such. In the old pre-Christian times we do not hear of this, though it was a sort of thing that would appeal strongly to the feelings and memory. No Sadducee seems to have suggested Catalepsy to discredit the real resurrections recorded in the Gospels,[2] and the fear of being accidentally buried alive is never alluded to by the classic writers, though so picturesque and so capable of poetical treatment. Any cases of apparent death that did occur were burnt, or buried, or otherwise put out of the way, and were never more heard of. But after the establishment of Christianity, tenderness, sometimes excessive, for the remains of departed friends took the place of the hard heathen selfishness. The dead were kept closer to the congregations of the living, as if to represent in material form the dogma of the communion of saints. This led to the discovery that some persons, indeed some persons of note (amongst others, Duns Scotus the theologian, at Cologne), had got out of their coffins, and died in a vain attempt to open the doors of their vaults.

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[1] Van Swieten's Commentary on Boerhaave, § 1040, vol. x. p. 170.

[2] Had he done so, it would have been a telling argument for the Council and the Scribes; but it would have small weight with an experienced physician now, nor would it make the miracle any the less in his eyes. He would know that it is quite as supernatural to detect a cataleptic in a funeral-train accidentally met at the city gate, or in one who had lain three days in a tomb completely rolled up in mummy cloth, as it is to raise the dead.

Others were more fortunate. Those who have visited the Lutheran cathedral at Magdeburg, have probably not failed to notice a quaint monument to the Frau von Asseburg. There is her effigy on it in stone, kneeling with her husband, and, in the style of the period, a goodly line of sons on one side and daughters on the other support the pair. The inscription relates how that this noble lady was, after her marriage, supposed to be dead, and placed in the family vault. Luckily the entrance was left unclosed that night, for she rose up, returned to her home and husband, and bore all this fair family after her strange experience of the tomb.

Such events caused no slight panic at the time, and probably led to the custom, still kept up in many parts of Germany, of fastening a bell-pull to the hand of a corpse when laid in the public mortuary. Some cases of resuscitated cataleptics have even occurred in modern times, according to the statement made by Archbishop Donnet to the French Senate last spring.

Catalepsy may be a premonitory symptom of other diseases. Epilepsy has already been mentioned. De Haen relates, in a clinical lecture, a case he saw of a child of twelve, who began by being cataleptic, and ended by reciting the metrical Protestant version of David's Psalms, saying her catechism with proof texts, and preaching a sermon on adultery.[1] And in several other stock cases, somnambulism seems to have been a complication. Marx saw a girl who became cataleptic from being frightened at a fire (like my patient at St. Mary's), and afterwards went out of her mind.[2] In Goebel's case of a young soldier, Catalepsy complicated the invasion of melancholia.[3] Sauvages says he saw an old man in the hospital at Alais, in whom Catalepsy alternated with quartan fever.[4]

Catalepsy seems to be sometimes voluntary, or at least capable of being brought on by very little external aid. Of this, St. Augustine gives an instance within his own knowledge:—

"There was a certain presbyter of the name of Restitutus in a parish of the diocese of Calami, who when he pleased (and he was often asked to do it by those who wished to have ocular demonstration of the strange fact), just by having a noise made like as of somebody crying, used to convey himself out of the influence of sensation, and lie like a corpse. So that not only was he insensible to people pinching and pricking him, but sometimes fire had been brought and he burnt with it, without any sense of pain, except from the wound afterwards. The body seemed to be motionless, not in consequence of any voluntary effort, but from want of sensation, as was made the more probable by the absence of any appearance of respiration, as in a dead body: yet people's voices, if they spoke out very clear, he said afterwards he could hear as if they were a long way off."[1]

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[1] De Haen, Ratio Medendi, vol. i. cap. xxxiii.

[2] Marx, De Spasmis, § 61.

[3] De Catalepsi, autore Theophilo Goebel. (A Berlin inaugural thesis, 1818.)

[4] Mémoires de l'Académie Royale des Sciences, &c. 1742, last page.

Persons liable to this form in various degrees lose, by yielding to it more and more, their power of voluntary control, so that exhibitions of it are easily brought on by others who assume an influence over them. They are told in a positive manner that they cannot raise their limbs, cannot open their eyes, cannot feel, and they really seem to lose temporarily motion, sight, sensation. Mesmer turned this artificial production of disease to profit (his own), and it has been largely experimented upon of late years. But the unfortunate subjects of it have brought to their masters so much "gain by their soothsaying," that deception has largely adulterated the real phenomena, and it is difficult to find a genuine patient. For this reason I thought it preferable to quote a case from a writer of unimpeachable shrewdness and honesty, and far from credulous, though destitute of the light of modern science, than to detail the experience of our own generation.

In the artificial disease and in the natural, somnambulism (*clairvoyance*) is a frequent complication, as appears from several cases cited by Tissot. (Œuvres, tom. xiii.)

It was not a groundless idea to suggest that, as we employ counter-irritants to relieve an internal unmanageable inflammation by one which is under our control and less injurious, so hysteria might be cured by inducing in its place an allied malady more subject to our will. But harm seems to be done by it, and probably only a limited number of the English race have a suitable diathesis.[2]

Both in the natural and artificial disease there is exerted a very different influence over the patient by different individuals. The sight of Dr. Jebb's face seems to have acted like the Gorgon's head in reducing his patient to instantaneous marble. My own experience is quite the reverse. Strange nervous phenomena always seem to be frightened away or subside into commonplace at my presence, and so perhaps my report of them is printed in less bright colours than the subject admits of.

Natural Catalepsy seems to become less frequent, or, at all events, the symptoms less marked and strange, as the world grows older. Some are even getting sceptical about its existence, and doubt the propriety of retaining it on our list of diseases. But even if it should be as extinct as the dodo or the great auk, this insult is uncalled for. The circumstances which surround the human race, especially when sick, are so altered, that it would be wonderful if some of the phenomena exhibited in pathological conditions were not altered too. Read the treatment adopted in many of the cases of Catalepsy quoted by the systematic writers. Take, for instance, that which M. Sauvages communicated to the Académie des Sciences,[1] where intermittent attacks of the disease occurred from time to time during several years. Though the patient was pale with a weak pulse, and though the blood could scarce be got to flow from the veins, yet she was bled, once from the arms, many times from the feet, and seven times from the neck. She had five or six repetitions of purgative medicine, not to mention *bouillons apéritives*, stomachic opiates, and twenty tepid baths, before they thought of giving her iron, which wrought a cure sooner than one could have expected. I lighted accidentally on another case communicated to the same scientific body by M. Imbert, in 1713.[2] It is that of the driver of the Rouen diligence, aged 45, who fell into a kind of soporific Catalepsy on hearing of the sudden death of a man he had quarrelled with. It appears that "M. Burette, under whose care he was at La Charité, made use of the most powerful assistances of art,—bleeding in the arm, the foot, the neck, emetics, purgatives, blisters, leeches," &c. At last somebody "threw him naked into cold water to surprise him." The effect surprised the doctors as much as the patient; it is related with evident wonder how that "he opened his eyes, looked stedfastly, but did not speak." His wife seems to have been a prudent woman, for a week afterwards she "carried him home, where he is at present: they give him no medicine; he speaks sensibly enough, and mends every day."

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[1] Augustine, De Civitate Dei, lib. xiv. cap. xxiv.

[2] Those who wish to pursue the subject of mesmerism will find it treated of with the broad views of a non-specialist by Feuchtersleben in his Medical Psychology, translated by the Sydenham Society.

Again the "*dame de Vesoul*," whom M. Tissot justly calls "*la cataleptique par excellence*," so characteristic were the phenomena, was attacked during Lent, when she had been starving herself in order to give alms to the poor, and was also at the same time worried by a lawsuit which had brought her to Besançon. Yet she was bled in the foot. Fortunately, after three days, her friends took her home to Vesoul. What happened then the reporter says was quite as wonderful as her illness,[3] namely, that she had no more medical treatment, and yet got well without a relapse. I cannot feel the same wonder, for I feel sure that the "powerful assistances of art"—bleeding, blistering, starving, purging, coddling, sympathising, and admiring—would have converted any of the cases under my charge into equally magnificent specimens of a long-continued intermittent disease.

But the fact of its being partly produced by art does not make Catalepsy a bit less of a reality, for the same may be said of all preventible diseases.

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[1] Mémoires de l'Académie Royale des Sciences, Année 1742, p. 409.

[2] Martin's Memoirs of the Academy of Sciences at Paris, vol. iv. p. 360.

[3] "Ce qui ne surprendra peut-être pas moins que sa maladie." (Tissot, Œuvres, tom. xiii. p. 16.)

Besides the effect of treatment, it is likely that the unrestrained manners and want of mental control peculiar to the barbarous ages of all nations, would render mediæval Europe liable to exaggerated exhibitions of all physical defects. And, as physical defects are indubitably hereditary, the national temperament would be thereby affected. As an example of what is alluded to, take one scene from early English history, and conceive it happening in the present day. Fancy four members of the Queen's Privy Council calling after lunch on a refractory archbishop who had voted against the Ministry that had appointed him, with the intention of showing him the error of his ways. Fancy them scolding and blaspheming "by God's wounds," giving him the lie, "jumping up and leaping about," "throwing about their arms," "twisting their gloves," "raving like madmen." Fancy him, red in the face, defying them, rushing after them to the door, calling one his lackey, and another "a pimp." Yet this is only a part of the want of restraint shown by both parties when Reginald Fitzurse, William de Tracy, Hugh de Morville, and Richard Brito called on Archbishop Becket on the afternoon of December 29th, 1170.[1] The mere fact of the murder, with the nauseous details of how Tracy picked out the brains with his sword, is not half so strange as such a scene. How many generations does it take to produce descendants of such men free from nervous disorders?

The deficient vitality of which Catalepsy is a manifestation occupies that puzzling part of the circle of life which lies between spirit and matter. We know so little about the chain which connects the two, that its links are reckoned by us as few and short, and we have no names for them. Yet when we see the varied phenomena produced by breaches in the connexion, we are led to feel our ignorance of the subject, and to conjecture that these abysses of incertitude veil a long list of vital functions.

In default of names for even the normal functions of this department of life, we must not expect an accurate nomenclature for their aberrations from health; and the most we can do in attempting to classify them, is to observe how near their origin lies to one or the other extremity of the series of vital acts which are interfered with—what relation their phenomena bear on the one hand to mind, and on the other to body. We shall thus have a natural order with pure insanity at the one end, and epilepsy traceable to organic lesion at the other. In the middle will lie ecstasy, Catalepsy, and hysteria, with many a blank between for the anonymous transitional forms. I do not think we can spare any of these names, and instead of clubbing them together, as some would fain do, under the common head of "hysteria," it would appear more useful to divide that disease, according as its emotional, anæsthetic, hyperæsthetic, or convulsive phenomena are most prominent. I say it would be useful to make a main point in each individual case whether the malady is most related to deviation from mental or bodily health; for I feel convinced it is only by this observation that we can avoid such disappointment as leads many to look upon hysteria, for example, as an *opprobrium medicinæ*, which makes them feel the same sort of anger against it as is roused by moral guiltiness, and disposes them rather to punish than to cure the patient who has thwarted them.

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[1] Hook's Archbishops of Canterbury, vol. ii. p. 497.

TREATMENT.—As to the treatment of Catalepsy, it is probable that valerian and ammonia, administered in draught or enema, whichever is most convenient, together with a modification of what cured the Rouenese stage-coachman, namely shower-baths, will accomplish all that is wanted in the way of medicine for the slighter cases likely to come under treatment in the present day. In longer continued cases Dr. Jebb's prescription of an opium plaster to the epigastrium, with tonics to the mucous membranes, is rational practice ; for Catalepsy seems to depend much on the mucous membrane of the stomach, as is the case with its sister malady, hysteria. But I would strongly urge upon all who have the charge of these and similar semi-mental, semi-corporeal manifestations, to take the hint given me by the second patient, and try to acquire (surely it is to be acquired by trying) the habit of command. Let them exercise it in the direction of supplying the deficient will, not of paralysing it, of demesmerizing instead of mesmerizing their patients, and it is astonishing how much pharmacopœial medication will be saved to both parties.

Catalepsy may be sometimes feigned. For its detection the most cruel means appear sometimes to have been adopted by our forefathers, such as burning, pinching, cutting, putting into coffins, and otherwise frightening the supposed impostors. A caution is therefore needed, that the trial of these methods would in England very properly subject the experimenter to legal proceedings, the more so as they are quite useless, and prove nothing. No malingerer could successfully feign the peculiar wax-like yielding resistance of a cataleptic muscle, and ought to be immediately detected by a medical man. If a doubt is felt, some expedient may be tried like that of Dr. Marx. Observing that really cataleptic limbs finally, though slowly, yield to the force of gravity and fall by their own weight, he attached a heavy body to the extended hand of a suspected impostor. She bore it up without moving; the intention of the experiment was explained, and she confessed her fraud.[1]

The points intended to be made concerning Catalepsy are these :—

1. That it is a rare pathological condition of mind and body, allied in its causes to hysteria, but not so apt to become chronic.
2. That it is not dangerous in itself, though it may be the precursor of dangerous disease.
3. That it may be artificially produced, but is not easy to feign.
4. That the treatment, moral and physical, should be conducted on the same principles as the treatment of hysteria.

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[1] Marx, De Spasmis. Halæ. 1765, § 19.

SOMNAMBULISM AND ALLIED STATES.

BY THOMAS KING CHAMBERS, M.D., F.R.C.P.

SOMNAMBULISM is a slumber so morbidly profound that resisting spontaneity is lost, and the obscure images, known as ordinary dreams, are able to exert a motor power. "Sleep-walking," where even the intricate concatenated motions necessary to preserve the body's balance are performed, is the most striking and dangerous exhibition of this state, and therefore has given a name to the disease; but it differs in degree only from sleep-talking, sleep-eating, and a form of nocturnal incontinence of urine and of spermatorrhœa. That it is not a partial waking is shown by the difficulty always found in fully waking a somnambulist, and also by the bewilderment and slow return of consciousness afterwards. This bewilderment, moreover, is often followed by headache and a clamminess of mouth, just like that of the condition known as "the intoxication of sleep" in those who have slumbered too heavily and too long. Again, decided somnambulists are entirely ignorant of what they have been doing during sleep: whereas dreams which occur during a partial waking are always remembered more or less. Again, the automatic acts done during partial waking are very short, have no continuity, and quickly end in a decided condition; whereas the acts of the somnambulist are consecutive one upon another. It seems impossible, therefore, to agree with Drs. Symonds, Hartmann, and others, who have regarded it as an incomplete sleep.

Still less can we agree with the superstitious awe which would represent it as an exalted state, in which the soul is freed from the trammels of the corruptible carcase. It is in truth a lower life, in which "the sceptre of reason is surrendered to a physically-directed fancy." (Feuchtersleben.) Instead of nearing the angels, man thus approaches temporarily the nature of ill-bred horses who refuse to lie down in their stalls, birds who roost standing on one leg, and gorged dogs who—

"Weary with the chase,
Lie stretch'd upon the rushy floor,
And urge, in dreams, the forest race
From Teviotstone to Eskdale Moor."

This morbid sleep usually arises in the first instance from eating too much. Perhaps the over-loaded stomach presses on the solar plexus, and produces a partial paralysis in the coats of the arteries, and so in the cerebral circulation. The explanation is the more probable, because sleeping with the head too low is another predisposing cause, whose action would be on the brain.

Strong mental emotion, excessive exertion of the intellect, violent grief, love, &c., probably act in the same way; namely, by arresting digestion and causing a weight at the stomach.

When, however, the habit is once established, it is persisted in even after the gluttony or emotion has been discontinued. In this it follows the rule of all morbid states of the nervous system, which are peculiarly apt to be retained in spite of the removal of their causes.

It is most common in youth, and at about the age of puberty. Then the sexes are equally subject to it; but later in life it seldomer attacks men than women.

Somnambulism is sometimes hereditary. A young lady, about whom I have been consulted a few times this summer, will often (sometimes two or three days a week) go off in the evening into a peculiar dreamy state. She talks and answers questions, though after an interval, walks about the house, goes to bed, remains quiet at night, and sometimes recovers her ordinary condition on waking: but sometimes her mother will go into her room and find her dressing in a vague, dreamy way. After a while she will stare, stretch herself like a person waking from sleep, and resume her natural lively manner. Her memory is always quite blank as to anything said, seen, or done during this condition. She has never had any hysteric or epileptic fits. Such are the symptoms, and her father asked me what name I should give to the disease. I hesitated at first, and then said that some might call it perhaps Catalepsy, but that the more proper name was Somnambulism. That was curious, he observed, for his mother had been afflicted with what was called both Catalepsy and Somnambulism, and he had heard it was in the family. I have also recently understood that a younger sister of my patient is falling into the same state, but I have not seen her. This lady had occasionally got out of bed when in her unconscious state, but it happened so seldom that no alarm on that score was expressed by the family.

Somnambulism is inconvenient to other people from the fright it causes, and dangerous to the patient from the awkward positions it puts him in when unprotected by reason. But it is by no means inconsistent with a fair condition of general health. It is, for instance, not unfrequent amongst boys and girls at school who bodily and mentally are quite equal to their companions.

At schools accidental accesses of it are liable to be fostered into a habit by the patient's room-fellows talking to them, and otherwise "drawing them out," when in this state.

Like epilepsy, and indeed all diseases of the nervous system, it is apt to become periodical. Some persons will walk, or talk, or wet their beds, &c. once a fortnight, week, or month, and so on with great regularity. That does not arise from an accumulation of secretion or excitability; for at first, and while the original cause is predominant, several attacks occur often close together in succession, and then cease. It is rather an evidence of the weakness becoming constitutional, after the original cause has been removed.

Somnambulism has in some rare cases alternated with catalepsy, of which M. Sauvages has recorded an instance. (*See* Catalepsy.) More generally, it alternates with a normal state.

There are cases recorded where the somnambulistic sopors have been so frequent and so long, that there is as much of a sleeping as there is of a waking condition, and thus has arisen the singular phenomenon called "double consciousness." Trains of thought are carried on from one attack to the next, though in the normal interval the mind is quite unconscious of them. A remarkable instance of this is recorded by Dr. Dewar, in the "Transactions of the Royal Society of Edinburgh," vol. ix. p. 365. A servant maid began by being subject to attacks of extreme sleepiness: then in these sleeps she began to be talkative. Soon there appeared more method in what she said: she personated an episcopal clergyman, went through the baptismal service for three children, and delivered an extempore prayer. Another time she was a jockey at Epsom, and rode round the kitchen on a stool. On awaking, all these pranks were quite forgotten, but in the succeeding fit she remembered all that occurred. Thus, one night a villain indecently assaulted her when somnambulistic. On the morrow the insult was forgot, but shortly afterwards she had a fresh attack and told her mother of it. She got well after an emetic and the return of the catamenia, which had been absent.

Dr. Abercrombie adds two more cases related to him by non-medical persons, and for that reason (probably) accompanied by more wonderful phenomena. (On the Intellectual Powers, Pt. III. sect. iv. § 2, II.) In principle these phenomena are quite in analogy with healthy dreams, which scarcely ever take cognizance of recent facts of the waking state. For example, in my own dreams, though I had the misfortune to lose a leg two years ago, I always seem to walk about as in youth. And I certainly remember a room-fellow at school who used to talk in his sleep on a class of subjects he never mentioned by day, and who seemed to recollect when in the same state next night that he had spoken of them before. But that an education should be carried on, and languages acquired, during somnambulism, as some strange stories record, is hard to credit. Possibly some confusion existed in the minds of the observers, and they mistook the waking for the sleeping state.

Somnambulistic phenomena have sometimes accompanied the artificial catalepsy of the mesmeric trance. They are called "*clairvoyance*," not that the patients see particularly clearly, but that the common sensorium is very receptive of those slight suggestions which it would neglect at other times, when its attention is occupied with the external world; and so they appear to careless observers to see with the tips of their fingers, the epigastrium, &c. when their eyes are closed. Singular exhibitions are thus produced. But with practice this rapid obedience to slight suggestion is soon acquired by even stupid people; so that jugglers have no difficulty in obtaining sham cases for shows, by no means easy of detection. This prevents the investigation of the subject by scientific persons.

TREATMENT.—1. The patient must be removed from the company of those who would be disposed to foster into a habit by experiment the recent establishment of the disease. This applies particularly to young persons at school, and those brought under the dominion of mesmerizers.

2. The patient must be prevented from falling into that morbidly deep sleep in which the special phenomena of the case are produced. This can be accomplished by waking them up once or twice in the night, before they begin to walk, talk, or do other unseemly acts. It may be done with great advantage during the second hour of sleep in cases of simple sleep-walking and of bed-wetting.

A young lady under my care who used to rise and make water on the floor without being aware of it, was relieved by this means.

M. Trousseau[1] knew a wealthy and beautiful girl, from whose feet wooers had been driven away by an incontinence of urine occurring nightly. At last the impediment to marriage was overcome by a hero. M. Trousseau cynically calls him "*un individu sans fortune*," implying that the girl's purse was her only attraction; but poetic justice requires a better motive for an act so richly rewarded; for, like the knight of whom the "Wif of Bath" tells, he found her "bothe faire and good," when expecting, like him, the latter only; the disgusting affliction vanished straight away—

> "And thus they live unto hir lives ende
> In parfit joye"—

at least they have the chance of doing so. Doubtless it was the prevention of over-profound sleep which cured her.

Some years ago a foolish young man from the country brought for my opinion an instrument which he had purchased of an advertising quack, designed to cure spermatorrhœa by compression of the urethra and prostate. The disease in his case, being purely imaginary, was of course incurable; but I should not wonder if the plan had been found useful, acting as an obstacle to morbid sleep. However, it would require careful medical superintendence.

A clergyman (who corresponded with me anonymously, and therefore I can say nothing of his general health) took by my advice, unsuccessfully, several remedies for spermatorrhœa, till he suspected that he abused himself during sleep; he tied his hands by a string to the bed-post, was awakened several times a night, and cured.

Another patient, troubled with really involuntary emissions, cured himself by having an alarum which he set so as to wake him occasionally in the night.

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[1] Clinique Médicale. Leçon LX.

Other attacks of spermatorrhœa I have found to take place during the abnormally heavy morning doze which lazy people indulge in after they have really had enough rest—the intoxication of repose. These patients should be told to get up and dress immediately after their first waking. It soon cures them.

3. Care should be taken that the head lie high in the bed, and that the body be not covered with too great a weight of clothes.

The son of an old and intimate friend of mine used to suffer when a child from incontinence of urine. Soon after puberty this inconvenience ceased, and has not returned. But at eighteen he has come to me complaining of seminal emissions, which have occurred on a few occasions more than once in a night. On inquiry, he said that on waking up after a defilement he had often found his head right under the bolster. He was advised to be careful in keeping a good hard pillow well down under the shoulders, and he has not suffered since.

4. Though prevention by means of keeping off too profound sleep is desirable, yet patients should not be wakened when walking, or in any other unnatural posture. They should be led back quietly to bed

> "Donec discussis redeant erroribus ad se."

Otherwise the fright is dangerous, especially to hysterical persons.

5. Light meals and digestible food are essential, and special expedients should not be trusted in till the general health has been brought up to the average.

SUN-STROKE.

By W. C. Maclean, M.D.

Definition.—A disease of the nervous system, excited by heat, sometimes following exposure to the direct rays of the sun, particularly when to heat is added the pressure of tight and unsuitable clothing and accoutrements, or both ; more frequently occurring when the above conditions combine with exhaustion, induced by great fatigue in hot weather, or from the effects of high temperature, night and day, on men breathing the vitiated air of crowded barracks or ships.

The affection is generally preceded by premonitory symptoms, such as thirst, heat, and dryness of skin, vertigo, congestion of the eyes, frequent desire to micturate, followed by syncope, often instantly fatal (the cardiac variety of Morehead), or by insensibility and stertorous breathing, with or without convulsions (the cerebro-spinal variety of the same author).

In both varieties the mortality is high, and unexampled congestion of the lungs is the most common morbid appearance observed after death.

Synonyms.—Insolation ; Sun-fever ; Coup de Soleil ; Calenture ; Heat-apoplexy ; Ictus solis ; Erythismus tropicus.

The first is the name by which the affection is designated in the official classification of diseases in use in the British army.

History.—Sun-stroke has been known and recognised as a dangerous disease from early times. Fatal examples of it are recorded by the sacred writers, and these have been referred to by most modern authors who have written on the subject. It is worthy of note that one of the blessings promised to those who shall be partakers of the better life that is to come, is, "that the sun shall not light upon them, *nor any heat*,"—a promise full of meaning to the inhabitants of the "dry and thirsty land" to whom it was first made.

Men of European birth who become sojourners in Hindostan are hardly more solicitous to protect their heads from the direct rays of the sun than are the various races who are children of the soil. In China, on the other hand, the inhabitants expose their closely shaven heads to the hottest sun with apparent impunity.[1] But when so doing they generally make vigorous use of their fans, as if they attached more importance to having a free current of air about their faces, than to protecting their heads from the sun's rays. Sportsmen in India constantly expose themselves in the hottest weather when in pursuit of game. Those who use reasonable precautions, who protect the head and spine by a head-dress adapted for the purpose, wear loose clothing of a suitable material, and abstain from stimulants, rarely suffer from Sun-stroke.[2]

On the other hand, as will be shown further on, men who are made to undergo fatigue under a hot sun, dressed as British soldiers used to be in such circumstances, in tight-fitting clothes, and encumbered with heavy and badly-adjusted accoutrements, wearing a head-dress which not only gave no protection, but concentrated the sun's rays on their heads, suffered from insolation in great numbers in a most fatal form.

Sun-stroke, if we are to judge from the older medical returns of the Indian army, was not a frequent or a fatal disease. Even in the eight years ending 1853-4, as appears from Dr. Hugh Macpherson's instructive analysis of later Bengal medical returns, only thirty-eight cases are recorded. This would be very puzzling if we did not know that a great many cases, which would now be entered without hesitation under the head of *insolatio*, were in those days "returned" under the heads of continued or remittent fevers; while those proving quickly fatal, with insensibility, convulsions, stertorous breathing, and such-like symptoms, were considered to be cases of cerebral apoplexy, and registered accordingly.

For example, in the case of the two wings of H.M. 13th Regiment, referred to by Martin, which marched, after some very ill-judged exposure and drilling in the sun, from Nuddea to Berampore in the midst of the hot weather, the men suffered terribly. As the result of one march, "the day closed with a sick-list of sixty-three, and eighteen deaths," all of which appear to have been registered as cases of apoplexy. It is certain from the description left by the medical officers, that the cases would in the present day be considered to have no pathological relation to apoplexy, yet Dr. Henderson was at a loss "whether to consider them cases of remittent fever or apoplexy." (Martin.) The symptoms were clearly those of insolation. Many other examples of a like kind could be given.

The following are some of the best historical instances of insolation occurring in the field and in barracks; they have been brought forward in more or less detail by nearly all recent authors on the subject, and for the last five years I have used them in my lectures in illustration of the different forms of this affection.

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[1] I have recently, however, seen an account of an epidemic of insolation, which attacked one of our trading ports in China, after many days and nights of unusually high temperature. The mortality was high, and by no means confined to the European community.

[2] Staff-surgeon Becker informs me that while on active service in China, a sudden order was given for a movement in the heat of the day. One commanding officer opened the canteen before the men marched; by way of precaution they partook freely of spirits. The effects of this injudicious measure were soon apparent: the men who thus indulged suffered twice as much from Sun-stroke as those who did not. On this occasion also it was noted that a large proportion of the victims had heart disease, in one or other of the forms I have elsewhere shown to be so common in the army.

In May 1834, the 68th Regiment, quartered in Fort St. George, Madras, attended the funeral of a General Officer. The regiment paraded in full dress at an early hour in the afternoon in one of the hottest months in the year, their tight-fitting coats buttoned up, their leather stocks, as stiff and unyielding as horse-collars, round their necks, heavy cross-belts so contrived as to interfere with every movement of the chest, heavy shakoes on their heads, made of black felt, mounted with brass ornaments with wide flat circular tops, ingeniously contrived to concentrate the sun's rays on the crown of the head, and without protection in the way of a depending flap for the nape of the neck. So dressed, the men marched for several miles. Before the funeral parade was over, the soldiers began to fall senseless—one died on the spot, two more in less than two hours. Men suffering from insolation in various degrees were brought into hospital all that night and part of next day. The cases that did not prove fatal, although their real nature was correctly understood by Dr. Russell, acting surgeon of the regiment, were all registered as cases either of continued or ephemeral fever. The symptoms in the fatal cases were thirst, excessive heat of skin, extreme prostration, immediately followed by gasping respiration, coma, stertor, lividity of the face, and death. After death no morbid appearance was found in the brain, but in the lungs of all there was extreme congestion. There lingers a tradition of this parade in Madras to this day.

The 63rd Regiment suffered in the same way, at the same place, and under circumstances precisely alike. (Martin: Influence of Tropical Climates.)

Of the next example the writer of this article was an eye-witness. The 98th Regiment joined the expeditionary force under Lord Gough in China in 1842. The regiment came from England in the *Belleisle*, an old 74-gun ship, and suffered from overcrowding. On the 21st of July, the 98th took part in the attack on Chin-Kiang-Foo, the final military operation of the war. The men were dressed precisely as already described in the case of the 68th Regiment. In this condition they had to take possession of a steep hill exposed to the fierce rays of the sun shining out of an unclouded sky. A great many men were struck down by the heat, about fifteen died on the spot, falling on their faces, as Dr. Parkes, on the authority of another eye-witness, has correctly described (Practical Hygiene); they gave a few convulsive gasps, and died before anything could be done for their relief.

The best history of an outbreak of insolation with which the writer is acquainted is that given by Dr. Barclay, of the 43rd Light Infantry, and published in the second number of the *Madras Quarterly Journal of Medical Science*. The 43rd Regiment performed one of the most extraordinary marches on record, having marched from Bangalore, in the Deccan, to Calpee, in Central India, a distance by the route taken considerably exceeding eleven hundred miles. The exigencies of the public service at that time (1857-58, memorable as the years of the mutiny in Bengal) were such, that this march, with the exception of a few brief halts at stations by the way, was made continuously, and a great portion of it was accomplished during the hottest season of the year. The men were exposed to a very high temperature by night as well as by day.

Dr. Barclay, while in a valley at the foot of the Bisramgunge Ghât, observed the thermometer at 118° Fahr. in the largest tents during the day, 127° in the smallest, and on one occasion he observed it at 105° at midnight. This prolonged exertion and continuous exposure to excessive heat by night as well as by day, exceeded the limits of human endurance. When they reached Nagode, "the indications of exhaustion in the altered looks of the men, their loss of flesh, and their evidently failing strength, were so obvious that they forced themselves on the observation of every one." But further on, on the march from Humeerpore to Calpee, Dr. Barclay records, "There was scarcely a man in the regiment whose strength was not reduced to a level with that of a child."

It is remarkable that no case of insolation occurred until the 28th of April, *i.e.* until the 43rd had marched 969 miles—until, in fact, the signs of exhaustion, first noted by Dr. Barclay, were apparent. From that date they increased in frequency. When at the foot of the pass, named above, cases "were brought to the hospital tents at every hour of the day and night, and although a large proportion of them recovered, two officers and eleven men were buried under one tree in the neighbourhood of the camp." (Dr. Barclay on the Natural History of Insolation.)

Boudin relates a terrible example of the effects of heat on a body of Belgian soldiers on the line of march, which may be fairly taken as a striking instance of the evil consequences of tight clothing and accoutrements under exertion in a high temperature. On the 8th of July, 1853, a body of men, 1,200 strong, marched from Beverloo to Hasselt. They started at eight o'clock in the morning. Only 500 reached Hasselt in the evening. Nineteen perished *en route*, and a great number in a state of furious delirium were taken to hospital. I do not think that anything so disastrous as this occurred during the unavoidable exposure of British soldiers to the fierce heat of the sun in Central India, in the years of the Mutiny, 1857-58. It is remarkable that the temperature on this occasion did not exceed 33° or 35° Centigrade. M. Boudin adds, that two well-known Egyptian astronomers, MM. Mahmoud and Ismaël, who were in Brussels on that day, assured M. Quetelet that they suffered as much from a temperature of 30·7° C. in that city as in Cairo under a temperature of nearly 50° C.: "*Nouvelle preuve de la nécessité de tenir compte de la qualité de la température.*"

But, as has been said, insolation occurs in barracks as well as in the field. The two best and most carefully observed examples of this form of the affection are those recorded by Dr. Butler, of the 3rd Light

Cavalry, at Meean Meer, and by Mr. Longmore, then surgeon of the 19th Regiment, stationed at Barrackpore, in Lower Bengal, and both published in the *Indian Annals of Medicine.*

Dr. Butler records that his men had not been overworked or fatigued, but at a time when the heat was excessive (102° in the shade) they were overcrowded. "Assuredly," says Dr. Butler, "those barracks most crowded, least ventilated, and worst provided with punkahs and other appliances to moderate excessive heat, furnished the greatest number of fatal cases."

Mr. Longmore's evidence on the same point is most important. Out of sixteen cases thirteen occurred in barracks or in hospital, and Mr. Longmore notes that one-third of his cases, and nearly half the deaths, "occurred in one company of the regiment quartered in the barrack which was manifestly the worst conditioned as to ventilation, and, indeed, in every sanitary requirement."

Mr. Longmore remarked also that "the patients seized in hospital were lying in two wards on the leeward side, and from circumstances of situation the warmest and most confined."

Insolation has frequently been observed on board ship, but almost always under conditions similar to those in barracks; that is, where overcrowding and impure air are added to the influence of excessive heat. Insolation is not uncommon on board the mail steamers in the Red Sea in the hot months of August and September; it has been observed that most of the cases occur while the sufferers are in the horizontal position in ill-ventilated cabins.

M. Bassier, Surgeon in the French Navy, reports (Dissertation sur la Calenture) that in the month of August 1823, the man-of-war brig *Le Lynx,* cruising off Cadiz, had eighteen cases of insolation, out of a crew of seventy-eight men. The heat was excessive ("33 *à* 35 *degrès Cent.,*" and much aggravated by calms. In this case the ship was overcrowded: "*le bâtiment, très petit, offrait peu d'espace pour le coucher de l'équipage.*"

M. Boudin (Statistiques Médicales) quotes from the same author the case of the French man-of-war *Duguesne.* This ship, while at Rio Janeiro, had a hundred cases of insolation, out of a crew of six hundred men. Most of the men were attacked, *not* when exposed to the direct heat of the sun, but at night when in the recumbent position—that is, when breathing not only a hot and suffocating, but also an impure air.

ETIOLOGY.—I have already remarked, that men will bear a high temperature in the open air with comparative impunity, provided (*a*) that it is not too long continued, (*b*) that the dress be reasonably adapted to the temperature, (*c*) that the free movement of the chest be not interfered with. As already remarked, British sportsmen in India often pursue their exciting amusement in the hottest weather; but as they are careful to dress suitably, they seldom suffer from insolation.

It will be remarked, that in all the examples of insolation in the direct rays of the sun given above, the sufferers were soldiers dressed and accoutred precisely as men ought not to be in the circumstances in which they were placed. Dress and accoutrements, then, are powerful aids to high temperature.

The case of the 43rd Light Infantry, as related by Dr. Barclay, brings out another predisposing cause, which appears to exercise a powerful influence, viz. exhaustion, the result of prolonged exertion.

This appears to act in various ways. First, there is a great waste of tissue, for a time,—that is, so long as the functions of the skin, lungs, bowels, and kidneys continue in tolerable activity, the blood is maintained in a state probably not far from its normal condition. But as exertion continues under a temperature seldom falling below 90° or 92° Fahr., and often reaching, as we have seen, 100°, 107°, and sometimes 118°, in a well-made tent, the function of the skin ceases, and the result of this must be not only the loss of the cooling effect of evaporation, but also blood impurity. Again, all observers note that under such circumstances, obstinate constipation of the bowels is a constant condition, still further promoting this impure condition of the blood. But not only may we reasonably suppose that the blood must be in an abnormal condition from the above causes; it is very imperfectly replenished by healthy, well-digested food. "The appetite," says Dr. Barclay, "gradually failed, and a feeling of nausea was generally complained of, the sight of food often exciting loathing."

In other instances there was nearly complete anorexia. It may be supposed that the activity of the kidneys may, to some extent, compensate for the lost function of the skin and the impaired eliminating action of other organs. But not to dilate on the fact that frequent micturition, although a common, is by no means an invariable symptom in the premonitory state of insolation, is it not possible that the inability to retain urine in the bladder is quite as much due to its quality as its quantity? "I cannot hold my water," was the almost invariable complaint of Dr. Barclay's men; and Mr. Longmore carefully noted the same thing in his cases.

Dr. Obernier is of opinion that although the secretion of urine is in excess at the beginning of a march, the quantity is lessened by prolonged exertion, doubtless because the blood has lost much of its water through profuse perspiration; and he states that suppression of urine often precedes Sun-stroke. "Now, suppression of urine means retention of urea in the blood," and accordingly in two cases of Sun-stroke Dr. Obernier found urea in excess of the normal quantity in the blood. Yet the same author declares that in his experiments on animals subjected to the effects of heat until they exhibited signs of Sun-stroke, he "could not discover a trace of urea in their blood."

If we look again to the cases quoted as occurring in barracks and ships, it will be seen that another cause besides heat was in operation. In all the examples given of insolation in barracks, the observers have noted the ill-ventilated, overcrowded condition of the places where the majority of the cases occurred. Many of the small bungalows occupied by officers in military stations in India, are quite as hot as any barrack-room, yet nothing is more rare than to see officers affected with this form of insolation. In the French ships, overcrowding and imperfect ventilation, with their necessary consequence, impure air, were noted by the surgeons who reported the cases.

It is then evident, from the above facts, and from many more of the same kind that might be adduced, that the pressure of tight and unsuitable clothes and accoutrements, excessive fatigue, with all its consequences, and the impure air of ill-ventilated barracks and ships, are powerful predisposing causes of insolation.

But it cannot be doubted that heat, and, speaking generally, heat long continued, is the true exciting cause of this formidable affection. The recently published observations of Dr. Obernier, of Bonn, put this opinion beyond doubt. Fick, a German physiologist of reputation, maintains that under the controlling influence of radiation and evaporation through the lungs and skin, the temperature of the blood in man is always the same.[1] But Obernier's experiments confirm the opinion urged in this article, viz. that if through any cause the cooling effect of the above processes be interrupted, "*as by warm and tight clothing*, by an elevated temperature of the outer air, by exposure to direct sunshine, or the overheated atmosphere of an engine-room, or even the forced deprivation of cold water; the effect is an accumulation of heat in the body, and an injurious if not fatal action of this heated blood on the nervous system, and through it on the heart."

The observations of physiologists have shown that the human body produces four times as much heat under considerable muscular exertion as during sleep. Obernier's exact experiments prove that the temperature of a man walking for half an hour increased by ½° Cent. or 0·9° Fahr. After two hours' walking in sunshine, his temperature rose 3°·6 Fahr. If exertion be continued in a hot atmosphere, or with the clothing so often adverted to in this article, or without the use of cold water, until the cooling processes of radiation and evaporation fail, "then the action of the heart grows weaker and weaker, the lesser circuit of the blood becomes overcharged, the venous vessels of the head grow turgid and sensibly expand; these symptoms develop gradually, but the resulting disease—Sun-stroke—makes its appearance suddenly." Obernier offers no opinion on the question whether or not nerve tissue undergoes any change of structure under heat. At this point the observations and experiments of Kühne, recorded in the second edition of Ludwig's Physiology, are full of interest.[1] This physiologist found that after exposing frogs to a high temperature, an electric current could with difficulty be transmitted along their nerves; at first it was lessened, and finally stopped altogether.

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[1] My friend Staff-surgeon Becker will shortly publish a series of most interesting observations on his own temperature on a voyage to India. Dr. Becker lived as much as possible on a uniform diet, which he weighed exactly, and ascertained the amount of urine excreted daily. He took his temperature with unfailing regularity eleven times in twenty-four hours. Every tenth day he fasted, and took his temperature every hour. The temperature of the external air was also carefully registered. The chart shows that the temperature of the observer rose or fell one degree with every twenty degrees of rise or fall in that of the external air. The disturbance on the days of fasting was very marked, the temperature invariably falling, and not recovering for many hours after food was resumed.

Other experiments by the same observer demonstrate that if the heat in any vertebrate animal exceeds 113° of Fahr., coagulation of the albuminous principle in the muscular system at once takes place.

There is no agreement among observers as to the effects of extreme dryness or moisture in increasing or diminishing the effects of heat. Insolation has been observed in both conditions. In the case of the 43rd Regiment, the hot, dry land winds were blowing. Mr. Longmore also notes the extreme dryness of the air at Barrackpore during the outbreak there; and in all the examples given, the disease disappeared with the first heavy fall of rain, attended with a rapid fall in temperature. On the other hand, Dr. Baxter, of the 93rd Highlanders, who gives four cases of Sun-stroke observed at Sealkote, considers that Sun-stroke is much more likely to occur when the atmosphere "is largely impregnated with watery vapour." (Dublin Quart. Journal of Med. Science, No. 81, Feb. 1866.)

Mr. Naylor also is of opinion that cloudy days, with "a moist condition of the atmosphere," favour the occurrence of insolation (Morehead's Clinical Researches). Exact observations on this point, with the wet and dry bulb, are much required.

It would appear that a hot and moist condition of the air is most favourable to the production of insolation in barracks, because not only does such a condition diminish the cooling effect of evaporation from the skin, but interferes with the artificial means used to reduce the temperature of the overheated rooms.

Duration.—The disease may prove fatal, as we have seen, in a few minutes, or the symptoms may last from one to forty-six or forty-eight hours.

Termination.—The disease terminates either in death or recovery, which may be complete or partial, certain sequels in a considerable number of cases appearing. These are persisting headache, the pain being either fixed or shifting; a chorea-like affection of the muscles, generally those of the forearm and hands; epilepsy, particularly in those who have suffered from this disease in youth, or who have a hereditary tendency to it. In some cases mental weakness, which may prove permanent, follows Sun-stroke. In one example, that of an officer of distinction, who lost his hunting-cap while pursuing a wild hog at speed, and in the eagerness of the chase rode for miles bare-headed, Sun-stroke was the result; from that hour his mind was affected, and complete recovery never took place.

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[1] Ludwig's Physiologie, vol. ii. p. 732.

SYMPTOMS.—Dr. Morehead has divided Insolation into three varieties,—the Cardiac, the Cerebro-spinal, and the Mixed. In the present state of knowledge this classification is useful, and it certainly appears to be founded on correct pathology.

In the Cardiac variety, although it is probable that the sufferer is himself conscious of some premonitory symptoms, there is seldom time for their full development so as to attract the attention of bystanders before the patient falls, gasps, and in some severe cases expires before there is time to do much, or anything, for his recovery, death taking place by syncope. This is the form most frequently seen in men exerting themselves in the heat of the sun when dressed and accoutred, as were the soldiers of the 98th Regiment at Chin-Kiang-Foo, or at the funeral parades at Madras above described.

In the so-called Cerebro-spinal cases, premonitory symptoms generally give notice of the coming danger. These are heat of skin—this is never absent; the heat is attended with extreme dryness, and is remarkably ardent and stinging, exceeding that of the worst form of remittent fever, which is sometimes as high as 107° Fahr.—giddiness, congestion of the eyes, extreme debility, nausea, and frequent desire to micturate. This last symptom is much insisted on by Longmore, and Dr. Barclay says that "I cannot hold my water" was often the first complaint made by many of his patients. It is a notable thing that headache is by no means a common symptom; it does not appear to have been complained of in a single instance in Dr. Barclay's cases.

Again, a wild shout of laughter, or an attempt to escape in terror from some imaginary enemy, sometimes precedes the more serious symptoms, to be presently described.

M. Bassier, in the case already referred to of the French man-of-war at Rio Janeiro, mentions that the utmost difficulty was experienced in preventing the men from throwing themselves into the sea :—"*Ils devenaient incohérents dans leur discours, poussaient des cris, menaçaient de geste et de regard, entraient en fureur, et semblaient mettre tous leurs soins à découvrir une issue qui leur permit de s'élancer à la mer.*"[1] On one occasion I saw a man in this condition suddenly possess himself of the arms of a sentry to defend himself from an imaginary enemy.

It is not by any means always that we have an opportunity of seeing the above premonitory symptoms. Where men in barracks are sensible of the approach of any of them, they generally assume the recumbent position, and in that state pass into a state of coma, the attention of their comrades being first called to their condition by their stertorous breathing.

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[1] Frenchmen under the influence of insolation seem strongly impelled to self-destruction.

Boudin relates that in 1836, "pendant une expédition du Général Bugeaud dans la province d'Oran, on a pu compter, en quelques heures, 11 suicides et 200 hommes atteints de congestion cérébrale, sur un colonne de quelques milliers d'hommes."

After a longer or shorter continuance of the above symptoms, the patient becomes insensible; the heat and dryness of the skin augment; the respiration becomes hurried, noisy, laboured; the pupils contract, and are quite insensible to light; the conjunctivæ become more congested, "pinky" (Barclay); the heart acts tumultuously; the pulse in men in ästhenic condition being at first rapid, but distinct, but as the case progresses unfavourably, becoming compressible, feeble, and irregular; convulsions are frequent, but not invariable: sometimes they appear early, in other cases they immediately precede death.

Dr. Barclay expressly says, that "in a large proportion of cases, from the commencement of the attack to its termination in death, the patient never moved a limb or even an eyelid."

In the Mixed form of Morehead, the symptoms partake of both varieties, and the fatal event is brought about partly by coma, partly by syncope.

DIAGNOSIS.—The diseases with which this affection appears to have been confounded are cerebral apoplexy, and various forms of fever, such as ardent continued fever, and even some of the graver forms of remittent. With the first named it has no pathological relations at all, and it is to be regretted that the term "apoplexy" continues to be appended to any of the names in use to distinguish this disease. In both apoplexy and the cerebro-spinal variety of Sun-stroke, there is coma; but the pulse in apoplexy is slow, generally full, sometimes intermitting. In Sun-stroke it is quick and sharp. In apoplexy the breathing is slow, irregular, and explosive in expiration; in Sun-stroke, it is rapid, noisy, but not explosive. In apoplexy the pupils are usually dilated, or one is more so than the other; in Sun-stroke both are contracted, and the conjunctivæ are deeply congested. The skin in apoplexy is not hot, and is often cold and moist; in Sun-stroke it is always, except in some rare examples of the cardiac variety, very hot and excessively dry.

Paralysis (hemiplegia) is the almost invariable result of cerebral hæmorrhage; it never follows immediately on Sun-stroke, and is rare even as a sequel of that affection.

From ardent continued fever, the premonitory symptoms already enumerated, more particularly the frequent micturition, and the early supervention of insensibility, will distinguish it. The above, with the history of the case, ought to suffice to distinguish it from the hot stage of remittent fever.

PATHOLOGY.—On one point, at least, all modern pathologists are agreed, viz. that the superheating of the blood which precedes and accompanies Sun-stroke, has a depressing and not a stimulating effect on the nervous centres; and as the general recognition of this has had a powerful effect in leading to a more rational form of treatment, it is a decided step in advance.

The opinion given by Dr. Morehead, that the sthenic constitution of the newly-arrived European predisposes to the cerebro-spinal variety of insolation, must, I venture to think, be taken with some qualification. Without doubt it was this variety that prevailed most in the 43rd Light Infantry; yet, from Dr. Barclay's description of the miserable condition to which his men were reduced before the disease appeared among them, they were in anything but a sthenic condition. On the other hand, the men of the 63rd Regiment at Madras, and the 98th at Chin-Kiang-Foo, fresh from Europe, were in the very opposite condition; yet it is clear, from the description given of the symptoms, that they suffered from the cardiac variety. It appears to me that the constriction of the men's chests by tight coats, the pressure of their unyielding stocks and accoutrements, had much to do in determining the particular symptoms—an opinion strengthened by all that I have learned from a careful study of the effects of dress and accoutrements on the organs of circulation and respiration, and by the revelations of the committee appointed to investigate this subject. At Chin-Kiang-Foo, the soldiers of the 18th Royal Irish, the 49th, and 55th Regiments were quite as much exposed to the sun as the 98th; they did not suffer from insolation, but they marched and fought without stocks, and with their jackets open.

I am also strongly impressed with the opinion that blood impurity, induced by the vitiated air of overcrowded barracks, tents, and ships, powerfully aids heat in bringing about that condition of the nervous centres which leads to the development of the terrible symptoms of insolation. Were it not so, the high temperature so often observed in the small houses and tents, particularly of many junior officers, would make this disease more common than it is among them.

MORBID ANATOMY.—The blood is invariably found in the same condition as after death from lightning, or blows on the epigastrium,—that is, fluid.

When death occurs so quickly as it generally does in this affection, there is really no time for much organic lesion; some congestion of the cerebral vessels is a common, but by no means an invariable, appearance.

The most common condition in all varieties, but more particularly in the cardiac, is congestion of the lungs, with distension of the right heart. This congestion is more complete in this than in any other disease.

MORTALITY.—If Sir Charles Napier is correct in the statement he makes in a letter published in his Life, that out of forty-four cases of Sun-stroke which occurred at Nussurpoor on the 15th of June, 1843, forty-three proved fatal, this is the highest mortality on record.

Dr. Barclay only included severe and well-marked cases in his table, and his death-rate was 42·734 per cent.

Dr. Butler's at Meean Meer was 43·3 per cent.

Dr. Morehead gives the following table :—

	Treated.	Deaths.
Mr. Hill's collected cases	504	259
Dr. Taylor's, Gazeepore	115	16
Mr. Longmore's, Barrackpore	16	7
Mr. Lofthouse, 14th Light Dragoons . .	80	10
Dr. Simpson, 71st Regiment	89	24
Mr. Ward, 3rd Bombay E. Regiment .	25	6
Mr. Ewing, 95th Regiment	60	17
Sir Hugh Rose and Dr. Stuart, 25th Regt.	200	—
Field Hospital, Hansi.	29	10

PROGNOSIS.—The most unfavourable signs are prolonged and complete insensibility, without movement (this is a much more unfavourable symptom than occasional convulsions); intense heat of skin, persisting notwithstanding the free use of the douche; increasing congestion of the eyes; tumultuous action of the heart; failing pulse; lividity of hands and feet.

If convulsions appear after such symptoms have been present for some time, they indicate the near approach of death.

PROPHYLAXIS.—1st, In barracks. The measure now about to be carried out in the construction of barracks in India, viz. having dormitories in which not more than from ten to fifteen men can be accommodated, will do more to diminish the frequency of barrack insolation than any single remedy with which I am acquainted.

Meanwhile, I cannot too strongly insist on the propriety of at once pitching a sufficient number of tents for dormitories, whenever the night temperature rises to 90°, so as to diminish, by at least one-half, the number of men in barracks during the night. The manner of cooling both tents and barracks is so well understood in India, that it is useless to insist upon it here.

In future, troops in India are never likely to be drilled in a hot sun, or paraded to attend military funerals in heavy marching order at 3 P.M. Even in that "military hothouse" Madras, where such customs "lingered long and latest died," it is to be hoped they will never more be heard of.

As a rule, save under the pressure of real military necessity, European troops are not likely to be moved during the hot weather; and where such a cruel necessity exists, the terrible lessons taught during the Mutiny are not likely to be forgotten.[1] When troops of necessity must march in hot weather, they should do so in as "open order" as military reasons will allow, and frequents halts should be called to allow the overheated bodies of the men to cool. This point is much insisted on by Marshal Ney, doubtless from observation of its necessity.

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[1] Dr. Obernier lays it down as a rule that when the thermometer in the shade marks from 86° to 88° Fahr. all marching of troops, if for practice only, ought to be avoided.

According to the same authority, a man engaged for an hour in foot-racing, the temperature of the air being 61° Fahr., experiences a rise in temperature of 5° even when perspiring profusely.

The necessity for light clothing, suitable protection for the head, neck, and spine, is now well understood and provided for by existing regulations.

At such times it will be good economy to engage an extra number of water-carriers, so that a never-failing supply shall be at hand, not only for drinking, but for *douching* purposes. I cordially assent to the practical suggestion urged by Dr. Barclay, that the hospital tents used on such occasions should be the best that can be made, and should be furnished with the best appliances, to maintain as low a temperature as possible. If patients suffering from insolation can quickly be brought into an hospital tent 15 or 20 degrees cooler than the one from whence he has been taken, his chance of life will be immediately increased. Nor is it necessary to say much on the good effects of temperance.

It is to be hoped that the day is not distant when the spirit ration will be a thing of the past.

TREATMENT.—In the days when insolation was commonly mistaken for cerebral apoplexy, the lancet was usually the first resource. The mortality even now, under a mode of treatment more in accordance with sound pathology, is often exceedingly high; but when blood-letting was the rule, recovery was the rare exception.

A few years ago, in an outburst of insolation on board one of the mail steamers in the Red Sea, this was the mode of treatment pursued. A fatal issue resulted in every case.

During active service in the presence of the enemy, an officer of rank had Sun-stroke. The assistant-surgeon in medical charge of the battery where this happened had the sufferer instantly removed to the nearest shade, stripped him, used the douche freely, and had the satisfaction to see his patient revive and consciousness return. An *official* superior, "*an older, not a better*" physician, unhappily coming up at this critical moment, insisted on opening a vein: a few ounces of blood trickled away, and so did the life of the officer. Mortal syncope immediately followed the operation.

It is needless to insist on this point, for, as Dr. Morehead has observed, "there is now great unanimity of opinion" on the treatment of Sun-stroke, and by universal consent the lancet has no place in it.

At the earliest possible moment let the sufferer be carried to the nearest shade, stripped, and assiduously douched with cold water over head, neck, and chest. If this be effectually and quickly done, the powerful impression on the cutaneous nerves will soon re-establish respiration, at first by gasps and catches, soon in a more regular and tranquil manner. It will also reduce the heat of skin. It may require to be done again and again; in hospital it may be necessary to envelope the patient in a wet sheet, and to ply the fan or punkah over him vigorously until the skin is reduced to a more natural temperature,—a measure, however, requiring to be done under medical supervision.

The patient should be encouraged to drink freely, and if vomiting follows, this will often aid in relieving the congestion of the lungs. The douche used as above described is a powerful remedy, and as Dr. Abercrombie long since pointed out, it may be abused, particularly if it is applied too long to the shaven head. Morehead has also well cautioned us against its prolonged use in a routine way, when the skin is cold and clammy, and the respiration sighing—under such circumstances we must restrict ourselves to dashing water over the face and chest. When the heat of the skin is excessive we may avail ourselves, if ice is at hand, of Dr. Parkes's suggestion, and give an enema of ice-cold water. We should also apply ammonia, with the usual caution, now and then to the nostrils. The bowels being always constipated, the sooner they are relieved the better, by the use of purgatives and enemata. If the skin refuse to act, even after the free use of the douche, and maintains its high temperature, a trial may be given to *Warburg's Tincture,* the most powerful sudorific with which I am acquainted.[1] The occurrence of moderate diarrhœa seems to favour recovery. Support and a judicious use of stimulants must not be neglected.

If sensibility be not restored and maintained by the douche, a blister should be applied at once to the nape, and, if need be, to the shaven head. There is much unanimity as to the good effects of this measure.

The late Sir James Simpson, of Edinburgh, long ago taught us how invaluable the inhalation of chloroform is in the convulsions of children depending on cerebral irritation. In India I have saved the lives of many by acting on this advice. Dr. Barclay in like manner found chloroform inhalation useful "in the convulsive form of the disease, attended with extreme nervous irritability,"—a class of cases in which, he adds, "the douche is inadmissible from the agony it occasions." In some instances life was saved by this remedy; in all it was prolonged.

TREATMENT OF THE SEQUELÆ OF SUN-STROKE.—Although by careful and judicious treatment many recover from the immediate effects of Sun-stroke, considerable numbers are incapacitated by it for service in India, or in any hot climate, without at least a more or less prolonged stay in a cold climate. This is precisely what we see after concussion. Out of the large number of cases of concussion I have seen in India from falls from horseback, I have hardly seen one make a complete recovery without a visit to Europe.

Persistent headache is one of the most common sequels to Sun-stroke.

At Netley during the invaliding season we are never without such cases, and very obstinate and intractable some of them are. When the pain is fixed, counter-irritation to the nape is recommended. I have seldom seen it of much use. I have seen this troublesome symptom follow Sun-stroke in this country, and be quite as intractable and obstinate as after insolation in India. A lady bathing at Cowes had a slight "stroke" of the sun; she did not lose consciousness. For more than a year after the occurrence this lady (who was under my observation) suffered from headache as severely as any of our invalids from India sent home after Sun-stroke in its gravest forms. Of late I have been more successful with the bromide of potassium than with any other remedy. It is far, however, from being a specific, often it fails entirely, as it did very notably in the case just recorded.

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[1] *Vide* article Remittent Fever, Vol. I. p. 608.

Great attention to the functions of the skin forms an essential part of the treatment in all the varieties of the sequelæ of Sun-stroke, for it is impaired in all. Frictions, bathing, exercise in the open air, are essential. When the headache is not fixed, but shifting, it will often be found to depend on a weak condition of the digestive organs, and careful treatment suited to the peculiar features of each individual case is required.

In epilepsy following Sun-stroke the prognosis is generally favourable. The fits usually subside on the patient being removed to a temperate climate.

ALCOHOLISM.

By Francis Edmund Anstie, M.D., F.R.C.P.

Definition.—A disease of the general nervous system, induced by continued excesses in the use of alcoholic liquors. It manifests itself usually in a chronic, but occasionally in an acute form. Its characteristic phenomena are muscular tremor and progressive muscular weakness, insomnia, hallucinations of sight and (less commonly) of hearing and smell, busy delirium, diminished or deranged intellectual and moral force, together with dyspepsia, slight jaundice and morning vomiting. In advanced cases there are also paralysis of sensation or motion, or both, convulsions, epilepsy, dementia, and general degeneration of the tissues of the body. Tendency to death slight when the original cause of the malady can be removed. The fatal result is either produced by exhaustion from protracted acute delirium, or slowly brought about by progressive degeneration of the nervous centres, or of some important organ of nutrition.

Synonyms.—Alcoholismus chronicus, Delirium tremens, Delirium potatorum, Mania potatorum, Ebrietas, Chronic alcoholic intoxication, Trunksucht, &c. &c. These various names obviously apply to various aspects of the disease accordingly as it occurs in the acute or the chronic forms, between which there was formerly no sufficient distinction made.

History.—The "history" of Alcoholism, to use the expression in its proper medical sense, is simply the story of the excesses of each individual patient as regards the daily allowance of alcohol, the duration of intemperate habits, and the kind of liquor taken, especially as regards its degree of concentration.

Etiology.—This part of the subject is extremely complex. The simplest portion of it is that which refers to the exciting causes, and it will be best to dispose of this first.

1. The *exciting causes* of Alcoholism can be better appreciated at the present time than formerly, because our increased knowledge of the physiological action of alcohol has enabled us to explode some errors of theory which were almost universal in medical writings and popular belief about the disease. The prime source of these errors

was the general tendency to notice only the more acute nervous affections which are caused by alcoholic excess—namely, delirium tremens, maniacal excitement, and terrifying hallucinations (horrors). These phenomena had been observed to occur frequently, and were believed to occur always in consequence of a temporary abstinence from drink after a course of excessive indulgence. This presumed sequence of cause and effect fitted exactly with the classical denomination of alcohol as a "stimulant," a member of an ideal class of medicaments which possessed the peculiar property of exciting vital function in such a manner that after a longer or shorter period a "recoil" was inevitable, under which the forces of life were reduced below their natural level. The great feebleness which was observed to characterise the acute delirium of drunkards was supposed to be due to this kind of exhaustion from the withdrawal of an accustomed stimulus.

At present our ideas are very different. In the first place it has been abundantly shown by various writers, of whom Ware[1] was the earliest, that abstinence from drink by no means always, or even most frequently, precedes the outbreak of delirium tremens or of alcoholic mania: on the contrary, these accidents commonly overtake the patient in the midst of his excesses. Secondly, alcohol, in doses which singly are capable of producing drunkenness, and frequently repeated may bring on acute delirium, &c., has been proved to be a true narcotic poison, of the same class as the so-called anæsthetics, chloroform, and sulphuric ether. Given in these large doses, its influence is entirely in the direction of paralysis—suspension of nervous activity; and this suspension of nervous activity (increased by other sources of deficient vital power, which we shall have to notice as incidental to the circumstances of the chronic drunkard) is itself a sufficient explanation of the nervous debility which brings about the delirious crisis. And thirdly, the modern researches which have enabled us clearly to identify a chronic alcoholic intoxication, often reaching over a period of many months or years, have revealed the fact that in the multitudes of instances the acute attack merely exhibits in full development symptoms which had been partially recognisable for a long time previously.

It may now be taken for certain that the phenomena of which we have to treat under the denomination of Alcoholism, are due in the first place to the direct action upon the nervous system of a blood-supply charged with a high percentage of alcohol. If we surround a living nerve (partially dissected from its connexions) with alcohol of a certain strength, we find that it becomes paralysed—*i.e.* incapable of transmitting impressions—through its affected part; while a very weak mixture of alcohol and water is incapable of producing this effect. Similarly, if an animal absorb into its circulation a certain quantity of alcohol within a given time, the nervous centres and the peripheral nerves become (though in less degree) paralysed. That this effect is at least in part, due to direct action of strong alcohol upon the nervous tissue can hardly be doubted, considering the analogy of the well-ascertained local effect in the above experiment: there is, however, a co-operative cause of no small importance; namely, it has been ascertained by the researches of various observers that the impregnation of the blood with large quantities of alcohol interferes with its absorption of oxygen; it thus becomes unfitted to support healthy nervous functions. Under these combined influences the nervous tissues, and particularly those of the central organs, become more and more unfitted for the performance of their proper functions: and this change progresses with a rapidity proportionate to the strength and frequency of the alcoholic influence. It is counteracted only by one circumstance—the elimination of portions of the alcohol from the system, which goes on by the medium of all the excreting glands, but more especially by the kidneys, the skin, and the lungs. Upon the activity of these organs in performing this task probably depends, *cæteris paribus*, the impunity of the drinker from the ill effects of the poison upon his nervous centres. Thus it comes to pass that the occupation and many surrounding circumstances of the drinker modify his symptoms in an important manner, as will be noticed more particularly under the head of Predisposing Causes.

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[1] Remarks on the History and Treatment of Delirium Tremens. Boston, 1831.

It is necessary here to recall the principal facts which are known with respect to the action of alcohol upon the organism. This substance is easily absorbed from the stomach, especially when that viscus is empty. If the dose be moderate and the administration well timed, the effect upon the nervous system is simply that of a restorative stimulant. Sensations of fatigue are dispelled, the mind works more freely, a healthy sense of warmth is diffused through the body, and the arterial system acquires an increased tonicity if it was hitherto deficient in that quality. The latter fact, which is due to the influence of the remedy upon the sympathetic nerves, is capable of being demonstrated in a very interesting and convincing manner. The sphygmograph of M. Marey has the power of accurately representing, by its registration of the pulse-wave, the degree of arterial tonicity present; and by this unfailing test it appears that the small vessels, when relaxed in a condition of fatigue, are brought, by a moderate dose of alcohol, to a proper tension, from which they suffer no recoil. If, on the contrary, the dose has been immoderate, or administered at a time when it was not required, the pulse-waves give a precisely opposite indication—that, namely, which proves that arterial relaxation has occurred; and simultaneously with this the pulse becomes abnormally quick. At the same time other symptoms of a paralytic nature are observed, confined in the first instance to the spinal nerves and to the fifth cranial nerve. The former show their weakness by the occurrence of slight feelings of numbness, and an impairment of muscular sense in the extremities; the latter indicates its affection by the occurrence of slight numbness of the lips. The vaso-motor fibres of the fifth nerve discover their partially palsied condition by flushing of the face, congestion of the conjunctivæ, and lachrymation. The cerebral hemispheres next give notice of the alcoholic influence by the occurrence of intellectual confusion, and the hypoglossi becoming simultaneously affected, the muscular movements of the tongue become difficult, and articulation is impeded. The further stages of drunkenness consist in more or less noisy or sentimental delirium, passing gradually into coma; palsy, more and more complete, of voluntary motion and sensation; the medulla oblongata is palsied, and breathing ceases; and, last of all, the organic nerves of the heart become incapable of performing their functions, and cardiac life ceases. During all this process the secreting glands are affected, but in varying degrees: the kidneys in particular are singularly little acted on in some cases, and very strikingly in others; and the diuresis, which is the result of the latter condition, is the best safeguard against fatal results, as it involves a large elimination of alcohol. MM. Lallemand, Duroy, and Perrin were the first observers who clearly proved the elimination of unchanged alcohol, and the phenomena appeared to them so striking that they concluded, too hastily, that the whole amount of any dose of alcohol taken into the system was thus eliminated by one or other secreting surface. The facts adduced by these authors do not, however, justify any such inference. I have repeated their observations with much care and especial attention to the dose administered—a point singularly neglected by MM. Lallemand, Duroy, and Perrin. The result of these observations is, that a moderate dose (*e.g.* a pint of light beer or a glass of sherry) produces very slight effects in the way of elimination, which last over a few hours only, and cannot be reasonably supposed to represent the elimination of more than a small fractional part of the alcohol imbibed. On the contrary, when a dose has been taken sufficient to produce more or less profound intoxication, alcohol is so copiously eliminated by skin, lungs, and kidneys, especially the latter, that there is some reason to think that as much as from a fourth to a third of the dose taken leaves the body in an unchanged condition within the course of forty-eight hours. Even in this instance, however, there is no sufficient reason to suppose that all the alcohol leaves the body in an unchanged form; indeed there are the strongest reasons for thinking the reverse. It is true that the intermediate compounds, between alcohol on the one side, and carbonic acid and water on the other, which would represent the stages of transformation of the former into the latter, have not yet been satisfactorily proved to exist in the organism after a dose of alcohol has been taken; but in truth nothing like an efficient search has yet been made for them. The researches of Lallemand show us that elimination may go on for a period of forty-eight hours, and my own experience appears to indicate with certainty that not a trace of elimination of unchanged alcohol can be detected at a later date than this. But this is entirely contrary to what we know of the behaviour of those poisonous substances which are wholly eliminated in an unchanged form, especially when, like alcohol, they are of a low diffusive power.

According to the analogy drawn from such cases, alcohol, were it entirely eliminated in an unchanged form, ought to be traceable by the delicate chromic acid test in all the secretions for a period not of two *days,* but of two *weeks* or more, from the time of its ingestion. Probabilities are therefore greatly against the total elimination of alcohol in an unchanged form, even from the chemical point of view; and to conclude this part of my subject, I may say that probabilities are converted into what most persons will be inclined to consider certainties, when the remarkable physiological influence of alcohol in supporting vital power is taken into consideration. But as I have dwelt fully on these matters elsewhere, it is unnecessary to repeat facts which would take up a great deal of space in the telling.[1]

The exciting causes of Alcoholism may be understood, then, to be the repeated direct action of blood *strongly* impregnated with alcohol on the tissue of the nervous centres and branches, rendering them physically incapable of the due performance of their functions, and the influence of an insufficiently oxygenated blood-supply consequent on a morbid condition of the blood-corpuscles.

2. The *predisposing causes* of the disease are much more complicated. They should be divided, in the first place, into (*a*) those which are occasional, and (*b*) those which are constant.

(*a*) The occasional predisposing causes include those external circumstances which expose persons to the temptation of drink, and those internal sensations, produced by temporary illness, which bias them in a similar direction, or, by weakening the nervous system, render the effects of drink more sensibly felt.

Occupation is an important influence. For instance, a large number of cases present themselves at the hospital which are directly traceable to the frequent presence of the temptation, as in the case of workmen at breweries and distilleries, and potmen and waiters at taverns. In a higher grade of life, public-house keepers and the clerks and travellers for wine and spirit merchants, are especially liable to Alcoholism. There is, however, by no means such a preponderance of cases due to this direct temptation as might be supposed. A very large number of patients come from the classes whose business exposes them greatly to the *inclemency of the weather:* thus cab-drivers, coal-porters (especially the workmen who lade the river barges), and hawkers are very commonly large drinkers; they very frequently become the subjects of Alcoholism, and would be still oftener affected in this way were it not for the assistance to elimination which their outdoor life renders. *Monotony* of occupation is also highly predisposing, especially when combined with much confinement in close rooms. Amongst occupations of this kind there are none which have furnished me with so many and such serious cases as the trades of shoemaker and barber. The want of active outdoor exercise, of course, represses elimination, and much increases the evil. I have seen few more desperate cases of Alcoholism than some which have occurred in barbers who have been habitually confined to miserably small shops, and at the same time have earned enough money to pay for a great deal of drink.

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[1] The reader is referred for ampler information to the papers of M. Baudot, in the *Union Médicale* for 1863, and to my work on "Stimulants and Narcotics" (London, 1864).

Since this article was written and in type I have had time to make new researches, which enable me greatly to strengthen my statement as to the comparatively trifling extent to which alcohol is eliminated from the body in an unchanged form. With the assistance of my friend and colleague, Dr. Dupré, I have made experiments on a large scale, and with increased care, which prove indisputably that when alcohol is taken in non-intoxicating doses, the total elimination, in the twenty-four hours, only amounts to a fraction (generally a small one) of a grain for the kidneys, and even smaller quantities for the lungs and skin. Even in cases of intoxication I now believe that a very much smaller proportion of unchanged alcohol is excreted than I had supposed, and enormously less than MM. Lallemand, Duroy, and Perrin had represented. The total period during which any elimination goes on is also proved to be much shorter than had been previously supposed.

By a curious coincidence, it happened that a German observer, Dr. Schulinus, was investigating the same question simultaneously with myself. His results, obtained from a very elaborate and careful series of experiments, closely correspond with my own. Dr. Schulinus' paper will be found in the *Arch. d. Heilkunde* for 1866. My own observations, which were made in ignorance of the German researches, are included in my lectures at the Royal College of Physicians, which appear in the *Lancet* of July, August, and September 1867.—F. E. A.

Depressing Mental Influences are powerful predisposers to drinking habits; and besides this, they directly increase the liability of the nervous system to be affected with symptoms of Alcoholism, in virtue of their weakening operation upon it. There is a vulgar notion that drink is the simple and uncomplicated cause of the greater number of *crimes* committed by the poor. The truth is, that, in recognising the indisputable fact that drunkenness is often followed by crime of a worse kind, people are apt to overlook large portions of the history of the criminal, and especially the wretched poverty in which he is usually reared. The demoralizing influence of this poverty is the central fact on which we ought to concentrate our attention; it is a common cause of general reckless behaviour, of which drunken habits are only a part, although they doubtless render the commission of fresh crimes more probable. The same recklessness of despair has often been seen to produce intemperance in drink, where poverty had no share in its origin. In the higher classes we not unfrequently see men who have failed in some cherished speculation, or women who have lost the only object which they cared about in life, take to drink with an almost insane vehemence, although they may never have shown any such tendencies before. It is not that there is any particular temptation in the taste of the drinks to which they have recourse, for it is a fact that even the most refined and delicate women, when they take to these practices, altogether neglect the really fine-flavoured alcoholic compounds: they do not drink wine, for instance, but brandy, or some equally coarse and strong spirit. In truth, it is an accident which leads them to select alcohol: under other circumstances they would take opium or hashish, or any other intoxicant which came conveniently to hand, or they would plunge into the indulgence of some special vice which promised them excitement. They merely wish for *oblivion.* And this is the very motive which drives the poor in many cases to drunkenness, and which simultaneously inclines them to commit other rash and criminal acts; the wish to escape, in any direction possible, from the hideous dulness of a life which is one monotonous pain. *Starvation*—actual severe deprivation of food—cannot be an active predisposing cause of drunkenness; for the opportunity of getting liquor is cut off by the extreme degree of poverty which brings about such a state of things: it is rather the sense of embarrassment and misery, consequent on the difficulty or impossibility of paying debts, that is common in the lowest ranks of the middle class, which prompts to drinking habits.

The influence of various forms of disease in predisposing patients to Alcoholism is twofold. In the first place, there are many conditions of chronic weakness and suffering which are susceptible of great relief, when they are at their worst period of aggravation, by the use of alcohol; and this fact, accidentally learned by the sufferer, is from ignorance often perverted. The diseases of this class which are the most important are the whole group of neuralgiæ, the depression and faintness attending the menstrual period of some women, and the debility and low spirits which often distress nursing mothers. Under the influence of such disorders patients are extremely apt to use alcoholic drinks recklessly, and the foundation of drunken habits is thus laid. This subject is so important that I may be excused for dwelling on it, at the risk of a digression, because it is scarcely possible in a very few words to convey my meaning without danger of being misunderstood. It is frequently charged upon those physicians who recommend alcoholic stimulants in disease, that they are encouraging patients to indulge in one of the strongest temptations to drunkenness. This accusation is entirely unjust, if applied to those who administer the remedy on scientific principles. I have endeavoured to show elsewhere, that it is the use of doses which are large enough or ill-timed enough to produce symptoms of *narcotism* that can alone implant in the patient that craving for drink which forces him against his will to indulge, with constantly increasing intemperance, in the abuse of alcoholic liquors. But the ignorance of the layman who attempts to regulate his own medicinal use of alcohol frequently leads him to confound two radically distinct modes of operation, by either of which this substance may be made to relieve pain and nervous depression or restlessness. The use of such moderate quantities as fall short of producing any, even the earliest, of the *intoxicative* symptoms which have been already described, while it frequently relieves the patient's distress, leaves no disastrous after-depression or craving, but simply restores the nervous system to its healthy state. But pain, and various other forms of *malaise*, may also be relieved by the use of true narcotic or intoxicant doses which paralyse the nervous system for a time; and it is this kind of temporary relief which involves depression, and a sense of craving for stimulants during the period in which the drinker is recovering from his narcotic stupefaction. It is this wasteful misuse of alcohol, in the absence of scientific knowledge, which becomes a predisposing cause of drunkenness in the case of patients affected with the class of maladies to which I have now referred.

One special variety of chronic temptation to drink, depending on temporary bodily conditions, deserves more notice than it has yet received, but can be only briefly indicated here. I refer to the influence of the sexual orgasm which distresses particular individuals of both sexes (but especially females) in whom the development of puberty takes place in a difficult or irregular manner. The symptoms with which young females are affected under these circumstances are commonly treated (as "hysterical") by various household remedies, which contain more or less alcohol, such as sal-volatile, eau-de-Cologne, and various warming tinctures. These remedies are often swallowed in most improper and unnecessary quantities; and there is too much reason to believe that in this way the foundation of secret drinking is not unfrequently laid.

(*b*) There is another kind of predisposing cause, which is *constant* in its operation, and which is probably at least as influential, both in producing alcoholic excess and in aggravating its ill effects, as any of those occasional causes which have been enumerated—viz. a peculiar inherited constitution of the nervous system. In the course of a large experience of Alcoholism among the hospital out-patients, I have been greatly struck with the number of drinkers who have informed me that their relations, either on the paternal or the maternal side, have also been given to drink. And a still larger number are found on inquiry to come of families in which some nervous disorders (especially insanity, epilepsy, and neuralgia) have been markedly prevalent. The doctrine of the hereditary transmission of a neurosis which, according to the special pressure of external circumstances, may take the form either of intellectual insanity, of impulsive emotional impulsiveness, combined with moral weakness, or, on the other hand, of convulsive or neuralgic affections, has been much insisted upon by recent alienist writers, and especially by Moreau in his very able treatise on "Psychologie Morbide." My own experience has led me to a firm conviction that particular causes of nervous degeneration affecting individuals do very frequently lead to the transmission, to the offspring of those persons, of an enfeebled nervous organization which renders them peculiarly liable to the severer neuroses, and which also makes them facile victims of the temptations to seek oblivion for their mental and bodily pains in narcotic indulgence. I believe that things often work in a vicious circle to this end; and that the nervous enfeeblement produced in an ancestor by great excesses in drink is reproduced in his various descendants with the effect of producing insanity in one, epilepsy in another, neuralgia in a third, alcoholic excesses in a fourth, and so on. Among the higher classes, where it is easier than in the case of the poor to obtain tolerably complete family histories extending over two or three generations, careful inquiry elicits facts of this kind with surprising frequency. So strong is the impression left on my mind by what I have observed in this direction, that I am inclined to believe that the great majority of the most inveterate and hopeless cases of alcoholic excess, among the higher classes, are produced by two factors, of which the least important is the circumstance of external momentary temptation in which the patient has been placed, while the more momentous and weighty cause is derived from an inherited nervous weakness which renders all kinds of bodily and mental trouble specially hard to be borne. It need hardly be remarked that, in this view of the case, the fatal rapidity with which habits of intemperance exaggerate themselves is only what might be expected, seeing that the nutrition of the nervous centres would be still further impaired by each successive indulgence in poisonous doses of alcohol, and the power of moral resistance to feelings of depression and misery would be proportionately weakened.

SYMPTOMS.—The symptoms of acute Alcoholism are in general well known, and there is little difficulty in understanding their access and the order of their succession. What is far less generally understood is the slighter and more chronic form of Alcoholism which, in the majority of cases, precedes by a considerable time the occurrence of the delirious affection. Accordingly, it will be well to commence with the description of this chronic disease, since its characters may be properly considered as representing the earlier stages of a great constitutional malady.

(A) *Symptoms of Chronic Alcoholism.*—It is upon the motor nervous system that the influence of chronic excess is first discernible in the largest number of cases. Of an extremely large number of patients who present themselves at the out-patient department of Westminster Hospital suffering from this disease, certainly more than two-thirds, upon careful analysis of their complaints, state that a muscular inquietude, which might or might not amount to actual tremor, was the first disagreeable symptom which they noticed. In cases of gradual access the affection at first may amount to no more than an inability to keep the limbs of the body still without a special effort of attention—the exercise of the will being sufficient to render the muscles perfectly steady. The degree of motor disturbance is distinct from and independent of the peculiar mental restlessness to be presently noticed as arising somewhat later, although the occurrence of the mental affection very much aggravates the tendency to involuntary movement. This distinction is noticeable in relation to the nocturnal state of the patients. Long before the occurrence of terrifying dreams, of nocturnal delirium, or of hallucinations—even before there is *conscious* nocturnal disturbance of the mind at all—the patient feels an inability to sleep which appears to depend on the condition of the motor nervous system. Repeatedly I have been assured by persons suffering from the slighter degrees of Alcoholism that they go to bed with a sense of at least average drowsiness, but an invincible disposition to turn restlessly from side to side in the bed entirely prevents them from getting any sleep.

It is not very often that a patient asks for advice at the early stage of the disease, which is represented by the presence of the above symptoms only. More commonly he does not come under medical notice till the motor disorder has reached a further stage; and his complaint is now, probably, that he suffers from persistent muscular tremor. This symptom develops itself first in the extremities. Magnus Huss declares that it always appears earliest in the hands; but it is probable that this is a mistake, for in a majority of the cases which have come under my care the lower extremities were first affected; while it is less easy to detect tremor of the lower than of the upper extremities, and the former often escapes notice for some time after its commencement. Huss notes correctly the fact that even in the stage of persistent alcoholic tremor the patients can at first, by a strong effort of the will, restrain their movements for a time, but on the cessation of the effort the tremulousness is ordinarily worse than ever. A very old and general observation is to the effect that the tremor of Alcoholism is almost invariably worse *in the morning*, and it has been usual to assign as the reason for this, that the accustomed stimulus of alcohol has been withheld for some hours. The statement is plausible, because it is the fact that a glass of beer, or wine, or brandy, taken under these circumstances, will at once diminish the unsteadiness of the muscles: but another fact may be mentioned which strongly opposes the theory—viz. that common foods, such as bread and milk, or broth (if the stomach be not too much irritated to digest them), will answer precisely the same purpose. In truth, the excessive morning tremor of the chronic toper is due chiefly to exhaustion from failure to get sleep. What sleep he has had has been of an unrefreshing kind, and a complete condition of nervous prostration naturally results, from which he can only be rallied by food or drink.

Coincidently with the establishment of persistent muscular tremor, and sometimes earlier than this, certain cerebral symptoms present themselves. One of the commonest of these is a buzzing or a rushing sound in the ears, which is frequently, though not always, accompanied with dull diffused headache. Vision is also affected, with varying degrees of severity, the most trifling symptom being the appearance of *muscæ volitantes*, or of "clouds" before the eyes. Flashes of light are a more serious phenomenon, and their occurrence at night, just before the patient drops into his first uneasy half-slumber, is frequently the immediate precursor of the more definite visual hallucinations. Momentary attacks of vertigo are common. By this time the peculiar alcoholic insomnia is fully developed in the great majority of cases: the patient tosses from side to side during nearly the whole night, getting only broken snatches of sleep, and these almost always attended with disturbing, and often with frightful dreams.

The mental condition is now usually such as to distress the patient and to impress the medical observer who sees the case for the first time. Its chief feature is the uncertainty of purpose which the sufferer displays: independently of any fixed delusion, or even of a distinct feeling of terror, there is a mental inquietude which makes it impossible for him to settle to any ordinary occupation, or to complete the tasks which he begins. To this is added either violent temper or a feeling of dread which may be vague and unaccountable, or (in bad cases) may arise from actual delusions, such as the belief that an enemy is constantly lying in wait to inflict an injury, &c. This sort of delusion is not to be confounded with another kind, which consists in a vivid apprehension by the patient that he is in danger of falling down a precipice even when he is walking on firm ground in broad daylight, and which seems to me to be connected with rapidly progressing impairment of muscular *co-ordination.* Cases which display the latter feature are commonly of a dangerous type, and, unless energetically treated, pass rapidly to a hopeless condition as to recovery. The sensation as described to me is not like that of ordinary vertigo, or of fainting; it resembles the disagreeable nightmare which everyone has experienced on first falling asleep after an indigestible supper, or, still more closely, the hideous feelings which some persons (myself among the number) suffer from under the action of a large dose of Indian hemp. But it is not usually found among the earlier symptoms of Alcoholism.

The above is a fair description of the nervous symptoms under which the patient commonly suffers when he first applies for relief. The disorders of common sensation which are frequently produced by alcoholic excess are, in my experience, usually later in their advent. When the patient comes under notice, he may present either of several conditions as regards his outward appearance. There is not often, at this early stage, any very great emaciation, even in the case of habitual spirit-drinkers; but there may be every degree of fatness, from the unwieldy bulk of the country publican, who chiefly fuddles himself with beer, to the slight frame of the London hairdresser, who too often makes away with two or three quarterns of gin or rum daily. It is a great mistake, however, to push so far, as is often done in descriptive works, the contrast between the respective influences of spirit-drinking and beer-drinking. The haggard wretches whose portraits Hogarth has drawn in his picture of "Gin Lane" are emaciated to that degree quite as much from utter want of all the comforts of life as from the direct influence of spirit-drinking; and, in fact, one sees, in the classes whose circumstances are a shade more easy, plenty of gin-drinkers who (living chiefly on gin) have a good allowance of fat, if not of muscle. The countenance of the drinker (whether of spirits or beer) usually presents two remarkable features in conjunction—viz. great flabbiness of the muscles of expression, and red, watery eyes; the conjunctivæ are also very generally more or less jaundiced. To this is often added redness of the nose and cheeks, and an eruption, resembling acne rosacea, around the nose and the mouth. On inquiry we learn that, besides the already-described nervous symptoms, the patient suffers from morning vomiting, or at least nausea. This is nearly always the case, but there may be any amount—or no amount—of general symptoms of gastric or intestinal irritation, except this one symptom; and the tongue, in correspondence with these variations, may be in nearly any state, from perfect cleanness and moistness to dry red glaziness or thick yellow furring: the latter is its more common condition, especially at the back part. The morning vomiting is in my opinion not a mere dyspeptic disorder, but a true part of the nervous phenomena of receding narcosis. One symptom, which it is not easy to explain, but which nearly always exists, even where there are no signs of dyspepsia, is a peculiar foul breath-smell, which it is impossible to describe, or to mistake when once it has been smelt. It is quite unlike the odour of the alcoholic liquor itself, and may be separately distinguished even when the latter is also present.

Considering the enormous quantities of spirituous liquors which are drunk by many of the patients who apply for relief from the consequences of chronic Alcoholism, it would be natural for the reader who holds the usual opinion as to the origin of cirrhosis of the liver to expect that serious symptoms, produced by the latter disorder, must often complicate cases of the former. The case, however, is far otherwise, in my own experience. Of an immense number of patients in whom the nervous disorder has been clearly identified, I have only seen thirteen cases in which the symptoms of cirrhotic disease called for any special treatment, although a certain degree of cirrhosis was doubtless present in many of the others; and I cannot avoid the conclusion, that some very powerful element, over and above the influence of alcoholic excess, is needed to produce the severe type of that disease. To a less, but still a remarkable extent, the same observation holds good for kidney diseases of the degenerative kind. With regard to these disorders, I am convinced that other depressing influences must bear a large share of the blame ordinarily attributed to alcohol. How is it possible to form any other opinion, when of the multitudes of drinkers whose kidneys must be daily traversed by blood containing large quantities of alcohol, so few present any characteristic change of the urine, or other recognisable symptoms of renal mischief? Be this as it may, it is certain that renal, and still more hepatic, complications are very rarely the source of serious embarrassment in the treatment of chronic Alcoholism of the ordinary type which is indicated by such a group of nervous symptoms as is above described.

Not to anticipate unduly what will have to be said under the head of Prognosis, it may be stated here that the form of the disease which we have so far considered, is decidedly curable, tending in fact to right itself on the simple adoption of a plan of complete abstinence from the exciting cause of the mischief, combined with a nourishing and supporting diet, unless in the rare instances where sundry complications, which may fairly be called *accidental*, happen to receive a dangerous development. These complications arise out of the local irritant action of the more concentrated alcoholic liquors on the gastro-intestinal mucous membrane or on the air-passages, and will now be described.

The irritant effects of alcohol on the alimentary canal are chiefly seen in the case of spirit-drinkers, and more particularly in those who drink spirits *neat*, or highly concentrated. Beer-drinkers do, indeed, often suffer from a simple form of dyspepsia, and there is little doubt that slow degenerative changes are usually set up in the stomachs of these patients; but, except in the case of enormous habitual excesses, the dyspepsia is a transient phenomenon which rapidly disappears on the adoption of a rigid plan of abstinence together with a simple medicinal treatment. The more concentrated alcohols, however, when used for any length of time, may set up a formidable irritation which produces intense congestion of the stomach or the intestines, or both: in short, a greater or less portion of the tract in which the radicles of the portal vein take their rise is subject to severe engorgement. Perhaps the most serious consequence of such an action is the occurrence, which we now and then witness, of *profuse hæmorrhage* from the stomach or bowels. According to what I have seen, this is rare. I have not met with a dozen cases of this kind altogether: two of these—one a case of hæmatemesis, and the other of intestinal hæmorrhage—occurred in the same week, in the practice of Westminster Hospital, quite lately. It is a frequent thing, however, for drinkers to be affected with hæmorrhoids, from which more or less bleeding takes place.

Great numbers even of the heaviest drinkers never develop any further *specific* symptoms of Alcoholism than those which have been already described, and their vicious habit, if it shortens their lives, does so chiefly by impairing their general nutrition, and thus rendering them less able to resist the attacks of intercurrent acute disease, and at the same time more predisposed to constitutional maladies, such as gout for instance, to which they may chance to have a hereditary bias. Others suffer from attacks of delirium tremens (to be presently described), once and again. But in many other drinkers the nervous symptoms, still preserving a more or less chronic type, assume a far more serious development; and we have now to speak of these more extreme developments of chronic Alcoholism.

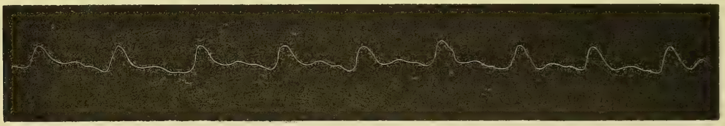
Of the earliest symptoms which indicate a dangerous degree of nervous degeneration, the occurrence of marked sensory paralysis is one of the most frequent. Unlike the corresponding affection of the motor nerves, sensory paralysis is most commonly exhibited in a slight degree in the upper extremities before it appears in the lower. The occurrence of any considerable degree of sensory palsy in the lower limbs is a sign of grave import: the patient so affected, unless he be induced at once to adopt a proper abstinence, and an appropriate medical treatment, is almost certain very quickly to experience some serious organic lesion of the brain. Simultaneously with the occurrence of a considerable degree of sensory paralysis, there is usually a great development of the muscular tremor, which, in several cases which I have seen, approached closely to the type of paralysis agitans. The mental powers are by this time usually affected in a marked degree—the most common mental condition being one of general intellectual enfeeblement and moral degradation, marked by cowardice and untruthfulness. At this point the progress of the case may diverge in either of several directions. In patients whose family history is strongly marked with the taint of insanity, a tendency to suicide is often developed, or else the sufferer sinks rapidly into a state of confirmed and incurable dementia. In others the function of muscular co-ordination is interfered with to a degree which makes the case resemble, at first sight, the affection known as Locomotor Ataxy. In others there occurs a sudden break down of nervous fibres in the corpora striata, or optic thalami, which produces a stroke of hemiplegic paralysis. In others, along with some symptoms of mental alienation, a general motor palsy is so distinctly observed as strongly to suggest the idea of commencing general paralysis of the insane. In others the rupture of a cerebral artery leads to an effusion of blood and the sudden occurrence of an attack resembling ordinary apoplexy. In others, again (but this is a very small class), the patient suffers attacks of convulsions indistinguishable from those of simple epilepsy. Epileptic attacks, occurring in this way, as a symptom of a very advanced stage of the nervous degeneration developed by chronic Alcoholism, are broadly distinguished, in a clinical and prognostic point of view, from the much commoner attacks of epilepsy in a subject known to be predisposed to or actually affected with that disease, as a mere consequence of a somewhat unusual alcoholic excess: the latter are of comparatively slight consequence, while the former indicate an altogether hopeless phase of alcoholic degeneration of the nervous centres. They are almost always accompanied by an advanced degree of dementia.

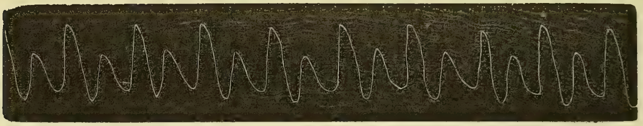
(B) *Symptoms of Acute Alcoholism.*—If we set aside the case of common drunkenness, as being rather an instance of narcotic poisoning, to be dealt with by toxicologists, than a morbid affection coming under the definition which we have placed at the head of this article, we may describe the symptoms of acute Alcoholism as presenting themselves under four principal forms—namely, Delirium Tremens, Acute Mania from drink, Acute Melancholia from drink, and Oinomania.

1. *Delirium Tremens.*—The clinical history of this disease was much misunderstood in former times. It used to be believed that in the majority of cases the delirious affection was produced, not by the direct poisonous action of alcohol upon the nervous system, but by the circumstance of an habitually intemperate person's leaving off the use of his accustomed potations. As a matter of fact it had frequently been observed that the sufferer from delirium tremens had ceased to

drink for one, two, or three days before the access of his more acute symptoms, and the exhaustion caused by the loss of his ordinary stimulant was supposed to produce those symptoms. Dr. Ware, of Boston (1831), was one of the first writers who pointed out that this statement includes a fallacy of observation. From an analysis of 100 cases, he proved that the cessation of drinking, where this occurs, is in fact produced by a feeling of revulsion to strong liquors, which is a part of the early symptoms of the acute disease in many cases; and, on the other hand, that very many patients do not leave off drinking at all, but the delirious attack supervenes in the midst of a debauch. This observation has been confirmed by Dr. Gairdner, and many other excellent recent writers, and at present the classical theory of exhaustion from withdrawal of an accustomed stimulus has but few upholders.

The first warning of the approach of delirium tremens is ordinarily given by the occurrence of complete insomnia. The patient may have long indulged to excess in drink, or he may be quite a novice in intemperance, but in any case a greater debauch than usual has commonly been perpetrated; and the sufferer finds himself quite unable to obtain any sleep, or at most can only gain short snatches of slumber, disturbed by horrifying dreams and visions: and during his waking moments, even in broad daylight, he suffers from hallucinations of sight which commonly take the form of disgusting or terrifying objects, such as snakes, insects, monsters, or of armed men pursuing him with threatening gestures. More rarely he hears voices denouncing threats, or mocking him: occasionally he experiences delusive sensations of disgusting smells. Often the occurrence of distinct visual hallucinations while the patient is awake is the first sign of the passage from chronic Alcoholism (which may have lasted for months or years, with a varying degree of insomnia, and perhaps with habitually distressing dreams) to the acute affection. During the first day or two days the patient is in an extraordinarily depressed state, with slow and feeble pulse, cold extremities, and a profuse sweating. The mental state is one of great anxiety, but there are usually no real *delusions:* even where visual hallucinations are present, the patient can by an effort of the will recognise them as such, and momentarily banish them from his sight. During all this time there is so complete an absence of appetite, in the great majority of cases, that no food, or scarcely any, is taken, and this circumstance probably mainly conduces to precipitate the onset of the second stage. In this the mere anxiety and nervousness is exchanged for incoherence of speech and wild excitability of manner, which sometimes takes the shape of causeless anger (though even then nearly always mixed with cowardice), and sometimes of great terror, which the sufferer often accounts for by pointing to imaginary terrific shapes which seem to people the room, and which he is constantly seeking to push aside with a restless motion of his hands. He talks incessantly, in a rambling fashion. Even when his terror or his anger is at its height he can generally be momentarily restrained by the influence of any onlooker who addresses him in a firm and determined manner, and may even be reasoned temporarily out of his hallucinatory imaginations. The pulse has now become quick (from 100 to 130 or 140 a minute): it is sometimes small and thready, sometimes soft and voluminous: but in every case which I have examined it gives a tracing, by the use of Marey's sphygmograph, in which the form of the pulse-waves closely resembles that which is observed in fevers and inflammations of a typhoid type, and is especially remarkable for the prominence of the phenomenon called "dicrotism." The annexed tracings will give a more accurate idea of the quality of the pulse than any description of the sensations which it communicates to the finger:—





Muscular *tremor*, which, from its striking prominence in many cases, has given the disease its name of *delirium tremens*, is by no means universally present. According to Craigie, whose observations on this point I believe to be correct, they are usually observed in the cases of confirmed dram-drinkers; and in many instances I have found on inquiry that they were only an exaggeration of a tremulousness of the extremities which had already existed for months or for years. But even when the characteristic tremulous movement of the arms and hands is not present there is a constant restlessness; the patient shifts constantly in the bed, and will get out of it twenty times in an hour if he be permitted to do so. The eyes are in almost constant movement; the pupils are usually, though by no means always, dilated. The temporal and carotid arteries throb violently in most cases; very often the face is flushed, but sometimes it remains deadly pale; nearly always there is much sweating, which is obviously due, in great part, to the constant muscular movements. The tongue is protruded, on the request of the physician, with an almost choreic jerk. It almost always trembles; usually it is covered with a yellowish fur, but it may be clean, red, and glassy on the one hand, or brown, dry, and cracked on the other.

It is usual to assign a limit to the second stage (which may last one, two, or several days) at the period when the patient first falls into continuous slumber; and no doubt the classical descriptions which assign this as the critical event to which convalescence may be expected to succeed, find a considerable superficial justification in clinical facts. But, in common with some of the most careful observers, I believe that to be a very erroneous and mischievous opinion which ascribes to a few hours' sleep anything like a distinctly curative power. It is true that in many, perhaps most, instances, the patient awakes, after his first sleep of considerable duration, in a condition of comparative convalescence. But, on the other hand, numerous cases have been observed in which the patient has sunk into profound slumber for many hours, and has awakened as delirious as ever, or in a state of complete prostration, which has rapidly terminated in death. Interesting considerations will be brought forward on this point, under the head of Prognosis, particularly with regard to the condition of the pulse, and the amount of success which has attended the efforts of the attendants to get the patient to take nourishment. In fact, the occurrence of sleep, even of considerable duration, marks with accuracy the commencement of convalescence only where we find the patient, on waking, clear in his intellect, free (or nearly so) from hallucinations, and with a pulse greatly reduced in frequency and yielding a sphygmographic trace such as will be presently described. The stage of convalescence once established, presents nothing particularly worthy of description. But instead of sleep occurring at all, the patient may pass from mere delirium into a comatose condition, with muttering delirium, eyes open, staring, and fixed, restless movements of the limbs more marked than ever, picking at the bed-clothes, or possibly profound stertorous coma, or violent convulsions, these symptoms being followed speedily by death. In other cases the patient, in the midst of violent delirium, with great excitability, suddenly collapses, as it were; the pulse becomes hurried, intermittent, and thready; the features pinched and ghastly, the breathing gasping, and death ensues in a minute or two, sometimes even in a few moments.

2. *Acute Mania* from drink presents symptoms which, though sometimes puzzlingly like those of simple delirium tremens, can usually be discriminated from the latter. The patient, who (invariably, as far as my experience goes) possesses some hereditary predisposition to insanity, is seized, in the midst of a drinking bout most commonly, with active maniacal delirium of a violent kind, and frequently displays a marked tendency to homicidal acts. In most of the cases which I have seen the whole aspect of the countenance and manner of the patient is different from that of delirium tremens, and there is comparatively little of the busy tremulousness of the hands so often seen in the latter disease. I believe that cases which are attended with positive intellectual *delusion* are nearly always of this, or else of the melancholic kind. The pulse, whatever its degree of apparent strength or weakness, as tested by the finger, is seldom so markedly *dicrotous* as in delirium tremens.

3. *Acute Melancholia* from drink presents the usual characteristics

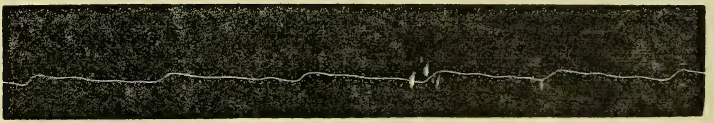
of melancholia from any other cause, but is marked by a special tendency to suicidal acts. The influence of a sound, protracted slumber, which in *mania* from drink is usually very beneficial, is far less so in *melancholia* from the same cause, as far as my limited experience goes.

4. *Oinomania.*—The fourth variety of acute Alcoholism is that curious affection which Roesch was the first to describe with precision, and which is now commonly called oinomania. It is, in truth, rather a variety of constitutional insanity than of alcoholic disease; but as the outbreaks owe many of their characteristic symptoms to the influence of drink, the disorder requires notice in a treatise on Alcoholism. The sufferers from oinomania are, I believe, usually descended of families in which insanity (and often insanity of the same type) is hereditary. Patients of this class very commonly, though not always, display their tendencies early in life; sometimes, indeed, on the very first occasion on which the opportunity for the free use of strong drink presents itself. It should be clearly understood that the term "monomania," which is often applied to the disease, very imperfectly describes the condition of the victims. Closer investigation of their mental state will usually discover the fact that they are liable to periodical recurrences of causeless exultation and bursts of self-confidence on trifling occasions; they then display great obstinacy, and a marked excitement of the animal passions generally: indeed the commencement of a drinking bout is often accidentally precipitated by the circumstances of temptation in which they are placed by loose company. Under the influence partly of an uncontrollable impulse, and partly of intoxication, they will perform truly insane acts; they take useless and purposeless journeys to remote places, or they lose their usual sense of decency, and expose themselves to disgrace by public acts of a degrading character. They exhibit symptoms which in many respects resemble those of simple delirium tremens, though there is usually a marked absence of that anxious terror which is almost always present in the latter complaint, and also a far less decided incapacity to sleep; indeed, there is sometimes very little insomnia. After lasting for a few days, a week, sometimes even a month or six weeks, the attack seems to wear itself out, as if rhythmically; and the patient generally recovers very rapidly his usual health, though he suffers "horrors" for a day or two. The condition of these patients in the intervals between these attacks is very different from that of the ordinary confirmed sot. Very often they live perfectly sober and chaste lives, and are even remarkable for active and intelligent management of their affairs. But this condition only lasts for two or three months, or six months, or at most a year, and then the old symptoms recur, and the patient is uncontrollably hurried into excesses of the most violent kind. Very rarely indeed is a sufferer from this disease really cured; it usually recurs with increasing frequency throughout life, and frequently ends in declared and permanent insanity.

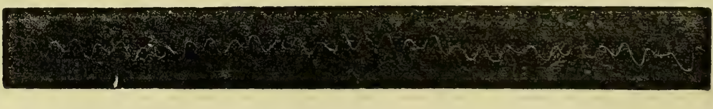
DIAGNOSIS.—The diagnosis of alcoholic diseases of the nervous system is not unfrequently surrounded with difficulties, especially in the case of the chronic forms. Chronic Alcoholism produces nervous symptoms which are particularly liable to be confounded with the following diseases:—1, Chiefly with commencing general paralysis; 2, with paralysis agitans; 3, with lead-poisoning; 4, with locomotor ataxy; 5, with hemiplegia or paraplegia from ordinary softening of the brain or spinal cord; 6, with epilepsy; 7, with senile dementia; 8, with hysteria; 9, with the nervous *malaise* associated with some forms of dyspepsia. The general group of leading symptoms whose presence enables us to affirm the diagnosis of chronic Alcoholism rather than that of any of these diseases is as follows:—The patient suffers from restlessness of mind (without delusions), insomnia, muscular fidgetiness, or actual tremor, morning vomiting; and presents flabby features, and watery eyes, and slight jaundice of the conjunctivæ. These symptoms make the diagnosis highly probable. If to them is added the occurrence of vertigo, muscæ volitantes, and terrifying dreams, it is greatly strengthened; and it is raised to the point of certainty, in my opinion, if there be also actual visual or auditory hallucinations in the form of visible shapes of men, beasts, &c., or audible voices. Indeed, the concurrence of distinct visual or auditory hallucination with only four other of the above-mentioned symptoms—viz. insomnia, morning vomiting, muscular tremor, and causeless mental restlessness—would of itself very nearly persuade me of the existence of alcoholic poisoning. Cases of commencing general paralysis (the most embarrassing counterfeits of the disease) may nearly always be distinguished by the presence of *mental exaltation*, the condition of the toper being uniformly one of mental depression, on the whole. The very rare cases of general palsy which do not display mental exaltation are wanting in the other features of Alcoholism, unless indeed when drink has been the exciting cause. As far as I have seen, chronic alcoholic poisoning *always* produces three or four of the leading symptoms which I have mentioned as specially diagnostic; and where an acne-like eruption of the face is also present, this settles it. The diagnosis of the acute forms of Alcoholism is usually far less difficult. We can generally get at a knowledge of the patient's mode of life in these cases; whereas the chronic toper is very commonly, especially if a woman, most cautiously and skilfully reticent and deceitful, and often conceals her habits even from her nearest relations. A case of considerable difficulty may arise in the distinction between delirium tremens and some forms of acute mania not caused by drink. The existence of delusions, not mere terrors, should bias us in favour of the diagnosis of mania, as should also the tendency to commit particular acts of violence, and especially lustful propensities; while the predominance of hallucination, especially when combined with terror of mind, tremor, and busy delirium, should predispose us to recognise delirium tremens. For the means of diagnosis between the different forms of acute Alcoholism the reader is referred to what has already been said under the heading of Symptoms.

Prognosis.—The prognosis in chronic Alcoholism, except in its more advanced forms, which are marked by the occurrence of serious paralytic or convulsive symptoms, or by considerable mental impairment, is highly favourable as regards recovery from the immediate symptoms. Mere abstinence, combined with simple but energetic treatment, to be presently described, will suffice in such cases to procure a rapid removal of all the unpleasant symptoms. Unfortunately, too many patients are biassed by long habit, by hereditary constitution, or by the dismally depressing circumstances of their daily life, in a way which renders their return to intemperance indefinitely probable. When once the more serious symptoms—such as paralysis, or epilepsy, or extreme and persistent muscular tremor—have occurred, cure, even for a time, is far more difficult, and the moral degradation of the patient, especially if a female, is so great as to allow small hope that abstinence will be observed.

In delirium tremens the main elements of prognosis are the occurrence or non-occurrence of sleep before the patient is very much exhausted, the condition of the pulse as tested by the sphygmograph, and the degree of success which attends the physician's efforts to get nourishment into the system. Sleep, as already remarked, is not of itself curative. The disease, in proportion to its original virulence, has a course of longer or shorter duration to run : this depends in great measure on the quantity of the poison taken, the sufficiency of the assimilative processes, the original strength of the constitution, and the degree in which it can be supported by well-assimilated food. Thus the prognosis is bad in the extreme when the dose of poison has been very large, the patient's constitution feeble, his powers of assimilation weak, and, in addition to this, disease of the glandular organs (especially of the kidneys) exists. Such a case is well-nigh hopeless. Almost equally bad is that in which any severe degree of *pneumonia* complicates the malady. The test, however, of the patient's chances which more than any other I am inclined to value, is the indications given by Marey's sphygmograph. In proportion as the pulse shows a tendency towards the normal form indicated by this tracing



are the chances good. On the contrary, such a pulse as the following



offers the extreme type of that typhoid form which is of most evil augury. This latter tracing was taken from a man, aged 40, who, after remaining for nearly a week in the delirious stage, fell into a sound sleep, which lasted for six or seven hours, and awoke apparently so much improved as to his nervous symptoms, that a somewhat confident opinion was pronounced in favour of his recovery. I augured the worst from the pulse-tracing; and in fact the patient sank rapidly, about twenty-four hours later. A somewhat extensive experience of this means of prognosis enables me to recommend it with much confidence. Mere rapidity of pulse counts as nothing in gravity, in my opinion, in comparison with the obstinate maintenance of the typhoid form of pulse-wave.

It is almost needless to remark that the circumstance of an attack being the first of the kind which the patient has suffered, renders it much less dangerous to life, as a general rule, than a second, a third, or a fourth would be; but there are important exceptions and qualifications to this law. Thus it may happen that a first attack of delirium tremens seizes a patient who has passed the line of middle age, and whose nervous system has been already much enfeebled by chronic disease or bad feeding, but who has never till recently indulged to excess in drink. Such an individual runs a great danger of sinking under the first acute attack; and the reason of this may be partly found in the feebleness of his system, and partly in the circumstance that his eliminating organs, especially his kidneys, have not become habituated to the irritation suddenly thrown upon them by blood containing large quantities of unchanged alcohol. The same embarrassment of eliminating organs suddenly charged with unaccustomed alcohol is doubtless the cause that a *young* man's first debauch (such as that of a young sailor, *e.g.* put on shore after his first voyage) so often causes an attack of delirium tremens; but here the constitutional strength usually enables the patient to bear up till the natural process of cure has time to be accomplished.

The prognosis both of acute mania and of acute melancholia from drink is decidedly good, at any rate on the occasion of first attacks, and provided that the affection is promptly treated. The probability of the case passing into one of confirmed insanity is of course progressively increased on the occasion of each successive acute attack.

The prognosis of oinomania is in one way very hopeful, in another almost entirely hopeless. The attacks of the acute affection may recur any number of times without any serious result: the patient, after a variable number of hours, days, or weeks, returns to his sober senses, and resumes his usual course of life. The hopelessness of the case lies in the taint of insanity which almost always lies at the foundation of the complaint, and which makes it almost impossible that the patient can effect a thorough reformation of his habits. However virtuous his intentions may be, and however strongly he may be urged by every consideration of prudence, or affection for those whose interests may depend upon his conduct, it appears as if he were impelled by a really irresistible force to yield himself, at certain intervals, to the temptation of drink. When the outbreaks become, as they usually do in the end, greatly more numerous than at first, there is reason to apprehend the speedy supervention of confirmed insanity.

COMPLICATIONS.—Of the complications of *chronic* Alcoholism it would be impossible to speak in detail, on account of their great number and variety. The only point to which I think it necessary to direct attention, is the question of the comparative liability of drinkers and of sober persons to phthisis. It appears certain, from the most careful statistics, and especially from those recently collected by Dr. Sutton, that the liability of drinkers to the ordinary forms of phthisis is considerably less than that of temperate people. On the other hand, every physician has now and then observed cases, which may be classed as "galloping consumption," which have occurred in persons who have been leading drunken lives, and which arrive with great rapidity at a fatal termination. I believe these victims of acute phthisis from drink are always descended of tuberculous families; and I think it likely that the starting-point of the actual tubercular deposit is to be found in continuous paralysis or semi-paralysis of the "nutritive" fibres contained in the pulmonary branches of the pneumogastric nerve, which is kept up by the patient's drinking habits.

Of acute Alcoholism, the only complication of which I shall separately speak is that of pneumonia. Nothing is more insidious than the occurrence of pneumonia in a subject whose nervous system is deeply poisoned with alcohol. A crucial instance of this occurred in the person of a patient who died in King's College Hospital many years ago, without its being suspected that anything more than delirium tremens was amiss, but whose right lung proved, on post-mortem examination, to be hepatised from apex to base. In this case there was no cough, no expectoration, no pain in the chest, and only so much frequency of breathing as seemed sufficiently accounted for by the restless muscular movements of the patient. It is most important, in every case of delirium tremens, that the chest should be periodically examined with care.

PATHOLOGY.—The pathology of Alcoholism naturally divides itself into three portions. The morbid influence which the poison exerts is of three kinds: in the first place, it acts as a local irritant (when highly concentrated) upon the mucous membrane of the stomach and the alimentary canal generally; and in the second place, after absorption, it affects the rate of movement and the vitality of the blood, and as a consequence of this impairs the nutrition of every organ of the body. And thirdly, it is clear that the nervous centres, independently of the ill effects on their nutrition of the blood-changes, have a certain chemical attraction for alcohol, which accordingly is found to accumulate in their tissues.

In the alimentary canal, and particularly in the stomach, the local effects of habitual large doses of concentrated alcohol are seen in permanent congestion of the blood-vessels, exaggerated or vitiated secretions from the gastric glands, and ultimately a degenerative change in the structure of the submucous tissues, which consists in the disappearance of characteristic secreting structures, and the hypertrophic exaggeration of fibrous tissue. Absorbed into the blood in large proportions, alcohol increases largely the amount of fatty matters in that fluid, and promotes congestion of certain important organs. The congestion of the lungs, liver, kidneys, &c. seems to be partly due to altered chemical relations between the blood and the tissues of those organs, and partly to a paralytic action of the alcohol upon the vaso-motor nervous system. It is by this latter action that I am inclined to account for the abnormal production of *sugar* in the liver, which has been experimentally observed by Bernard and Harley to follow the introduction of concentrated alcohol into the portal vein, and also for a largely increased excretion of water from the kidneys, which is one of the most invariable consequences of large doses of alcoholic liquors. It is indeed doubtful whether the degenerative changes which result from prolonged alcoholic poisoning are not in great part due to the direct chemical influence of alcohol upon the nervous tissues. The characteristic changes which have been observed in the brain, medulla oblongata, &c. of confirmed drinkers, consist essentially of a peculiar atrophic modification, by which the true elements of nervous tissue are partially removed, the total mass of nervous matter wastes, serous fluid is effused into the ventricles and the arachnoid, while simultaneously there is a marked development of fibrous tissue, granular fat, and other elements which belong to a low order of vitalised products. Essentially similar changes are observed in the lungs, the liver, the kidneys, the heart, and the larger arteries, which (after the nervous centres) are the most frequently affected. The cranial bones are also thickened by a deposit which is not of the nature of a true hypertrophy, for the bones lose much of their original texture, and become dense, almost porcellanous. There is much in these changes which reminds us forcibly of the effects on nutrition of tissues produced experimentally by Schiff and Mantegazza by the section of compound nerves, such as the fifth cranial, and the sciatic and crural of the lower limb; and suggests the idea that in alcoholic poisoning the starting-point (or at least one starting-point) of degenerative tissue-changes may consist in paralysis of those nervous branches which preside specially over nutrition, the distinct character of which has been so well pointed out by Brown-Séquard.[1]

It is highly probable, however, that a considerable portion of the degenerative influence of the continued excessive ingestion of alcohol is due to a chemical interference with the natural course of oxidation of the blood and tissues. Notwithstanding all that has been urged in favour of the view that alcohol is not transformed within the body, the balance of evidence is strongly in favour of the belief that a considerable portion of every dose of alcohol which is ingested does undergo oxidation in the system, and that to the diversion from its ordinary purposes of the inspired oxygen must be ascribed the diminished activity of elimination of carbonic acid, of urea, of chlorine, and of the acids and bases of the urine, which undoubtedly does occur in the subjects of alcoholic poisoning.

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[1] *Vide* Lanceraux, Archives Gén., Oct. 1865, for a full account of the Morbid Anatomy of Alcoholism.

TREATMENT.—1. The treatment of the chronic form of Alcoholism varies according to the stage of the disease which has been reached. In that large majority of the cases which come under our notice, in which the patient merely complains of nervousness, of inability to sleep, of muscular tremor, and perhaps of the slighter forms of visual hallucination, together with some dyspepsia and with morning vomiting, the treatment required is extremely simple. One has only to insure that the patient practises a proper abstinence from drink—to insist upon his taking a diet as rich in nitrogenous matters as may be, but at the same time such as his digestive system can appropriate—and to administer certain tonic medicines; and in nearly every case we may count upon a rapid disappearance of the unpleasant symptoms of which he has complained. With regard to the first item, the prescription of abstinence from drink, a good deal of difficulty may arise, and there is room for difference of opinion as to the expedient course. I wish to express the decided opinion that complete abstinence may always be carried out without any immediate danger to life or health, if proper care be taken to substitute a substantially nourishing diet. The danger of pursuing this course is not a physical but a moral one: all kinds of pledges which, as it were, *bind* the individual, have a tendency to lessen the force of such notions of personal responsibility as he may retain; he is apt to rest his confidence on the oath or formal resolution which he has taken, instead of teaching himself the virtue of self-restraint, as he would have to do if he were to accustom himself to the moderate use of alcoholic liquors. This is a question, however, which must be left to the practitioner's judgment in each case. The administration of a highly animalized diet is often a matter of difficulty at first, owing to the feebleness of the digestive powers, which renders the use of solid meat impossible, and even that of soups very difficult. Under these circumstances the greatest possible benefit may be derived from the administration of some of the better so-called "concentrated" preparations of meat, more especially Gillon's beef-juice, and a solid extract from this which is prepared by Messrs. Bell, of Oxford Street, as also the better specimens of the extractum carnis of Liebig. Without entering into the vexed question of the exact nutritive value of these preparations, there can be no doubt that they are powerfully reviving to an exhausted nervous system, and that simultaneously with the general improvement which they produce, the digestive organs become strengthened to deal with more bulky forms of animal food. The direct medicinal treatment of chronic Alcoholism in its milder forms is very simple. The presence of dyspeptic symptoms, unless they are very aggravated, and there is reason to believe that serious organic changes in the abdominal viscera have taken place, ought not to distract our attention from the main object of fortifying the nervous system; for with the observance of a proper abstinence from their exciting cause they will rapidly subside. The nervous tonic in which, after a great many trials of different remedies, I have come to repose the greatest confidence, is *quinine* in one-grain doses two or three times a day. It should be given from the very first, if possible; and this may be done, even when the stomach is very irritable, by administering the remedy in effervescence, with bicarbonate of potash and citric acid. The symptoms which most of all distress the patient, in the majority of cases, are the persistent wakefulness and the tendency to visual hallucinations or to appearances of black specks, flashes of fire, &c. before the eyes: the insomnia is also, of course, a great obstacle to that repair of the nervous energy without which recovery is impossible. But it would be a mistake to suppose that soporific narcotics, in doses which in a comparatively healthy patient would produce a stupefying effect, are well adapted to relieve this wakefulness: on the contrary, they generally aggravate the nocturnal restlessness, besides seriously impairing the general health. Nothing has been more marked, in my experience, than the superior efficacy of direct tonics, and especially of quinine, in producing that nervous tranquillity which makes sleep possible. When these medicines prove insufficient, I have found a remedy, which has been recommended by several authors, very useful—namely, sulphuric ether, either given in half-drachm doses three times a day, or a single dose of one drachm at bedtime. A good addition to such a night-draught is half a drachm of tincture of sumbul.

Another remedy, which has proved very successful in the hands of my friend and late colleague, Dr. Marcet, is the oxide of zinc, which, according to that author, has a powerful effect in inducing sleep. He recommends it to be used at first in doses of two grains twice daily, but this quantity may be progressively increased, if necessary, until ten, twenty, thirty grains daily, or even larger quantities, are taken. I have given this medicine very patient trials, both in the smaller and in the larger doses, and I cannot say that I have been so favourably impressed by its action; and on the whole I am inclined to think that in the majority of cases quinine acts much more satisfactorily. It must also be borne in mind, as Dr. Marcet himself admits, that in certain subjects, especially the anæmic and the chlorotic, the continued administration of zinc is observed to produce a prejudicially depressing effect on the constitution. Nevertheless there is no doubt that oxide of zinc occasionally proves a valuable remedy. I think it should not be administered in larger quantities than at most six grains daily; and I concur with Dr. Marcet in the recommendation that it should be given shortly after a meal, as it otherwise sometimes occasions nausea.

A much more effective remedy than zinc appears to be the bromide of potassium in ten or twenty grain doses three times a day. Although I have not yet had the opportunity of trying this medicine so extensively as I should wish, the results obtained have been very good. In several instances it has at once removed distressing wakefulness, dreams, and visual hallucinations. It is occasionally impossible to give this drug, however, from its exciting gastric irritation.

Now and then we find that sleep is not to be obtained by any of the remedies above mentioned, and we are driven to the use of some of the more recognised hypnotics. Of these one of the most effectual is the extract of Indian hemp; it should be given in small doses; from a quarter to half a grain of a good extract is quite sufficient, and a larger quantity is more likely to do harm than good to the majority of patients. Opium, if given at all, should be administered in the form of morphia, hypodermically injected; one-tenth to one-quarter of a grain is sufficient. But a medicine which is quite as effectual in many cases is good bottled stout given in one single dose of half a pint at bed-time.

In the more advanced cases of chronic Alcoholism, where the nervous centres are undergoing serious degenerative changes, as evidenced by the occurrence of paralysis, epileptiform convulsions, or grave mental deterioration, further remedial measures are required. Of these the two which have yielded me by far the most satisfactory results are cod-liver oil, and phosphorus in the form of the hypophosphites of soda or lime. Cod-liver oil, to be really of use, must be continued in tolerably full doses over a long period. Employed for so long a time as three or six months without intermission, I have seen it produce striking benefit even in advanced stages; and in some instances where it failed to produce anything like a cure, it caused great amendment of the most serious symptoms. The hypophosphites in five and ten grain doses, three times a day, have been particularly valuable, in my hands, in the treatment of cases which were distinguished by commencing paralysis of sensation. In one case which was marked by epileptic convulsions, with much impairment of the mental faculties, the combined use of cod-liver oil and bromide of potassium produced very beneficial effects. Another class of cases, those in which the predominant symptom is a very considerable degree of muscular tremor, are often greatly benefited by strychnia. Very small doses only are to be used; it is well to commence with the $\frac{1}{48}$th of a grain, and increase this to not more than the $\frac{1}{32}$nd of a grain, three times daily. Doses much larger than this have invariably seemed to do decided harm, especially increasing the tendency to vertigo, visual hallucinations, and noises in the ears.

2. The treatment of Acute Alcoholism. (*a*) Delirium tremens is a malady the treatment of which has experienced several changes correspondingly with the progress of accurate clinical observation. In former times—indeed a very few years since—the notion universally prevailed that the delirious symptoms were owing to the exhaustion

which was chiefly kept up by want of sleep; and, consequently, that the production of continuous sleep for several hours was the sole and all-important means of cure. It was therefore the custom to ply the patients with larger and larger successive doses of opium, with a view of drowning the delirium in narcotic stupor. Great mischief arose from this wide-spread belief and practice. In the first place, it has often happened that the patient, without ever sleeping at all, has passed first into a condition of coma-vigil, next of stertorous breathing, and at last sunk, fairly poisoned with opium. Again, a fact which was disregarded by the earlier authorities was this, that, without exerting any poisonous action upon the centres of consciousness, opium occasionally spends almost the whole of its depressing force upon the visceral nerves. A minor consequence of neglecting this fact was, that the patient's chance of assimilating food was often entirely ruined by the paralysing action of the drug upon the digestive organs: a much more serious one was the accident which has doubtless often happened, and which occurred in cases within my knowledge—namely, the rapid induction of a cardiac paralysis, the patient (without any cerebral signs of poisoning whatever) suddenly becoming ghastly pale, the pulse fluttering and coming to a standstill within a few moments. One such example was particularly striking, as it immediately followed two large doses of opium, which had been given in the vain hope of procuring sleep; the second dose was equally inefficacious as a soporific with the first, but its deadly effect upon the circulation could not be mistaken.

The idea that patients in delirium tremens require to be narcotised into a state of repose, may now be said to be abandoned by those best qualified to speak on the subject. In truth, the condition of the brain requires that sort of treatment which shall fortify and stimulate its functions. I have already argued at length, in another work, that every stimulant, when given in such restricted doses as alone deserve that name, is a promoter, but not an exhauster, of function, and that the idea of any depressive *recoil* following its action is purely fictitious. There are, accordingly, a great number of remedies of which the larger doses are narcotic, and the smaller stimulant, which in the latter form are capable of giving more or less relief to the symptoms of delirium tremens. It is not worth while to enumerate all these. The typical member of the group of stimulants is simple, easily-digested food; and the successful treatment of delirium tremens, in nine cases out of ten, depends on the regular and continuous supply of suitable nutriment, whereby the functions of the nervous system are supported during the struggle towards recovery. The principal kinds of food which are desirable are milk, soup, or strong broth with bread in it (and given *very hot*), the concentrated meat-foods already recommended under the head of Chronic Alcoholism, and raw eggs beaten up. The necessity for the administration of some nutriment of this kind is imperative; and if the stomach be at first too irritable or the anorexia too complete to allow of feeding by the stomach, it must be given in the form of enemata, so as not to lose a day, nor even a few hours.

It should be observed, however, that there are two classes of patients, in one of whom it is, and in the other it is not, desirable to employ some preparatory treatment of an *eliminative* kind. The value of *purgatives* has been recognised by many writers. They are eminently suitable to those cases in which a young and somewhat robust person has brought on delirium by drinking a very large quantity of spirits; in such instances a dose or two of medicine, producing free watery evacuations, effects a wonderful improvement (no doubt by ridding the alimentary canal of much of the alcohol which it has taken in). Where the strength of the patient is sufficient to allow of this plan being safely carried out, it will be found that the subsequent assimilation of food is rendered more easy and rapid, and that the stage of convalescence is comparatively soon attained. But in debilitated subjects it is far better not to attempt any forced increase of the eliminative processes, but to commence at once with the administration of the more easily digested foods in small quantities frequently repeated. The irritation of the stomach may be combated by the administration of ice, and of small quantities of soda-water and other aërated drinks, and one of the best modes of commencing the necessary feeding is by administering milk, mixed with one-third its bulk of lime-water, at frequent intervals. Everything is to be hoped for a patient who has been well supported by food from the early stages of the attack.

Of late years an important question has been raised concerning the therapeutic value of digitalis in delirium tremens. The practice, introduced by Mr. Jones of Jersey, of administering very large doses of tincture of digitalis (from half an ounce to an ounce and even more), was a startling innovation on the traditional practice in the use of this drug; such doses having formerly been universally regarded as dangerously poisonous, and calculated to produce fatal depression of the circulation. It has been proved, beyond doubt, that in a large number of cases these doses are at least harmless, and the testimony of a good many observers has now apparently established the fact that the delirium may frequently be quieted, and sleep obtained, by the employment of digitalis in this manner. It must be owned, however, that the question still remains in a very unsatisfactory position. The great majority of the cases have been treated with the *tincture*, and not with any simple preparation of digitalis: that is to say, the patients have, in fact, received half-ounce or ounce doses of *proof spirit* over and above the drug intended to act upon the disease. But it is well known that alcohol, in common with all the stimulant class of remedies, has often a beneficial influence in states of low delirium. In the presence of the very conflicting statements on the action of digitalis which have been published by different writers, I have endeavoured to clear the matter up by employing a strong *infusion* instead of the tincture; but it is unfortunately impossible, in many cases, to get the patients to take the remedy in this shape, and I have thus been hindered from effectively carrying out the experiment. The powder, given in pills, would be a better form. From the observation of a few cases treated with digitalis, in one form or another, which have been under my own treatment or that of friends, I have been led to the provisional conclusion, that in all probability a large number of the reported successful cases have either been instances of a spontaneous favourable termination of the disease, or have been slightly helped towards their happy issue by the alcohol which is contained in the tincture ordinarily employed. This consideration leads us naturally to consider the very important question —whether alcoholic liquors should or should not be used in the treatment of delirium tremens.

I am inclined to think that the moral argument has great weight here. In all cases, and more especially in first attacks, the subjects of which, we may hope, are not irremediably debased by drunken habits, it appears to be incumbent on us to use the time of sickness as an opportunity for possible reformation, unless alcohol were *necessary.* It would, therefore, seem to be our duty to commence the work by giving the patient's system an entire rest from the action of alcohol during the period for which he is under our authoritative guidance. In young subjects, therefore, and in first attacks, it is proper to abstain altogether from the use of alcohol. It is more difficult to carry out this plan with older patients, and with those who have been for a long time accustomed to depend upon strong drinks for a large part of their ordinary nutrition. In every case, however, I think it is our duty to abstain as long as possible from the use of alcohol, and before resorting to a treatment of such doubtful propriety, we ought to try less harmful narcotic stimulants. Opium and Indian hemp fulfil the indications which we require, under these circumstances, better than any others of their class. Opium *should never be administered by the stomach,* but always in the form of morphia hypodermically injected, in the dose of one-tenth to one-fourth or one-half grain. Where there is any reason, from the quality of the pulse, to believe that the circulation is much enfeebled, Indian hemp, in doses of a quarter to half a grain of good extract, is a less objectionable remedy, and I have seen it produce excellent effects.

A very important question is the propriety or otherwise of employing the inhalation of chloroform, in order to quiet the patient sufficiently to enable him to sleep: on this matter there has been the greatest difference of opinion. My own experience of this remedy may be summed up as follows:—In the first place, I have known from personal friends of two cases (and many others have been recorded) in which the patient died suddenly, from cardiac palsy, while the inhalation was proceeding. Secondly, I do not believe, though I have frequently tried it, that the action of small doses of a weak atmosphere of chloroform (such as would be free from the danger of producing cardiac palsy) is sufficient to induce sleep, or even to greatly reduce the patient's agitation, in the majority of cases. And lastly, remembering how few persons possess a high degree of skill in exactly graduating the dose of chloroform-vapour, it appears undesirable that it should come into general use in delirium tremens. For it is certain that the evil effects of a narcotic depression of the heart's action are much more serious in the case of this disease than of many other complaints. Given internally, in doses of twenty to thirty minims (or an equivalent amount of chloric ether), chloroform is less dangerous, but, as far as my experience goes, not more successful. Other practitioners, however, have met with more success in its use, and some have pushed it to much larger doses; but considering that forty-five minims taken internally by a healthy man has been known to produce full anæsthesia (though this is usually too little to produce such an effect), it is not advisable to run the risk of larger doses than I have named.

In all probability another remedy, which has only lately become the subject of attention in respect to delirium tremens, will prove one of the best of all the auxiliary means for quieting nervous agitation and hastening the advent of convalescence. I refer to the bromide of potassium.

In twenty-grain doses repeated every two hours the bromide succeeds, in a large number of cases, in calming the nervous agitation and procuring really refreshing sleep; it should be pushed till as much as two drachms have been taken in consecutive doses, if sleep is not procured before; but very commonly not more than three or four doses are required. As soon as the patient wakes out of sleep the administration should be resumed. My own experience is now sufficient to assure me that this treatment is incomparably more effective as well as more safe than the use of opium. It is more especially fitted for young and vigorous patients however, and especially to those who, while preserving considerable muscular power, have so injured their nervous centres by large excesses as to induce epileptiform tendencies.

A second remedy has lately been discovered, which appears to me to exactly fill the place of an appropriate remedy for those cases for which the bromide is not suitable—I mean the hydrate of chloral. Given in twenty-grain doses repeated at an hour's interval, chloral appears to me to act in a manner superior to that of any drug which has been used in delirium tremens. It is rarely that more than three doses are required to produce calm and refreshing sleep.

A remedy which has been used with great success in many cases, and with most unfortunate results in others, is tartar emetic. The handling of this drug in delirium tremens is an extremely difficult thing, for it requires much judgment to decide whether the constitutional strength of the patient is sufficient to support its undoubtedly depressing effects. I venture to believe that the directions, so often given, to employ antimony in cases which are distinguished by "active" delirium, with a bold and threatening (instead of a timid) expression of countenance, congested conjunctivæ, &c. are quite worthless. Such symptoms afford no measure of the patient's real strength, nor are they any warrant for the use of antimony; for this remedy must be given in considerable doses, if it is to do any real good: from a quarter to half a grain should be given three or four times, at intervals of one or two hours. When a favourable effect is produced, it is always accompanied (and I believe caused) by an increased secretion from the kidneys, or by profuse sweating, by which probably the elimination of alcohol is favoured. Scantiness of either or both these secretions is therefore the true indication for antimony. But it is necessary, even when these indications exist, to form a very accurate judgment of the strength of the circulation, and this, if we trust to the finger's estimate of the radial pulse, is most difficult. Fortunately, the use of Marey's sphygmograph will enable us to form a far more correct opinion than was formerly possible on this point. The symptoms which indicate a dangerous action of tartar emetic are faintness, *cold* sweating, and intermittence or irregularity of the pulse: the latter symptom should be carefully looked for with the help of the sphygmograph, which may detect it when the finger could not. If the first dose produces even a slight irregularity of cardiac rhythm, the medicine should be at once suspended. I wish to express the decided opinion that bromide of potassium and chloral are practically the only drugs we need ever employ in delirium tremens.

The treatment of the *complications* of delirium tremens hardly requires any special remark, except perhaps as to the complication of *pneumonia*. It is of course necessary, as a general rule, to be specially careful to avoid unnecessarily depressing treatment of affections the original cause of which is the action of a depressing narcotic poison such as alcohol: but this rule is of twofold importance in the case of pneumonia supervening in acute Alcoholism. I am satisfied that I have seen the life of a patient sacrificed by the administration of two or three consecutive quarter-grain doses of tartar emetic, under the idea that this treatment was specifically indicated by the affection of the lung. Tartar emetic, blood-letting both general and local, purgatives, and every other depressing treatment, are to be utterly proscribed in alcoholic pneumonia, an affection which is attended with much greater debility, especially of the heart, than its superficial symptoms would appear to indicate. The sphygmograph is very useful as a test of the real condition of things.

One important branch of the treatment remains to be briefly noticed. It is in all cases most highly desirable that a skilled attendant should be procured, and in cases where the patient is at once violent and of considerable strength, two trained persons, with experience of the treatment of lunatics, should be placed in constant attendance. It is scarcely necessary to say that the utmost violence of a patient should never induce us to employ bandages or the strait-waistcoat, if it be anyhow possible to secure sufficient nursing assistance.

(*b*) The treatment of acute mania from drink is a subject which belongs properly to the department of mental diseases, and

(*c*) The treatment of alcoholic melancholia is in the same position, as is also

(*d*) The treatment of oinomania.

In order to give as much continuity as possible to my description of the diseases grouped under the term "Alcoholism," I have purposely avoided long digressions upon the views held by other writers, and have made comparatively few quotations of their writings. But in order that the reader may have an opportunity of comparing this article with the teachings of other modern writers, I subjoin the following list of the principal works which are now looked upon as possessing authority on this subject :—

Sutton, Tracts on Delir. Trem. &c. : London, 1813. Roesch, Papers in Ann. d'Hygiène, t. xx. 1838. Rayer, Mémoire sur le Delir. Trem. : Paris, 1819. Ware, John, Remarks on the History and Treatment of Delir. Trem. : Boston, 1831. Peddie, Dr. J., On the Pathol. of Delir. Trem. and its Treatment without Stimulants or Opiates : Edinburgh, 1854 (pamphlet). Laycock, Dr., Pathology and Treatment of Delir. Trem.: Edin. Med. Journ. vol. iv. 1858-9. Huss, Magnus, Chronische Alkohols-Krankheit (German Edit.) : Leipzig, 1852. Marcet, Dr., On Chronic Alcoholic Intoxication, Second Edition : London, 1863. Carpenter, Dr., Use and Abuse of Alcoholic Liquors : London, 1850. Various papers by Dr. G. Johnson, in the *Lancet*.

VERTIGO.

By J. Spence Ramskill, M.D.

Definition.—The sensation of moving, or the appearance of moving objects, without any real existence of movement.

Description.—Vertigo may present two forms: in the one the patient complains of giddiness in himself, external objects remaining stationary; in the other external objects assume various abnormal positions: for example, articles of furniture in the room, or patterns of paper on a wall, seem to chase each other round the apartment; or, in rarer cases, the vehicles in the street appear upside down, or the pavement undulates, or feels elastic. On attempting to walk; the patient may feel himself drawn or impelled forwards, sidewards, or backwards, and he can only prevent himself obeying the impulse by a strong effort of volition. Minor degrees of disturbed balance, and the commonest sense of uncertainty of gait demand the same exercise of volition, for there is in all cases a perpetual fear of falling down or of rude contact against other persons or against surrounding objects. In slight cases Vertigo occurs only on movement; in severe ones, when at rest also, and even during sleep.

With both forms of Vertigo we occasionally find perversions of the special senses. Patients complain of mistiness of vision, of being unable to see more than half an object, or of one half being out of all proportion to the other half, of exaggeration in size of an object, of deafness, or of hyperæsthesia of the sense of hearing, the noise of passing vehicles assuming the intensity of thunder, or of metæsthesia or a perverted sense, ordinary loud sounds appearing clear, but soft and distant. In a distinct variety of Vertigo there is real deafness of one or both ears.

Associated with these functional disorders there are complaints of tinnitus aurium, a noise of pumping water, of intermittent pulsations of fluids, of the hissing of a tea-kettle, of the noise of machinery, in fact of many kinds of noises which defy and escape description; most commonly the noises are permanent, although they may vary in intensity whilst the Vertigo is intermittent, yet the noises are loudest during the vertiginous attack.

PROGNOSIS.—It may be taken as a rule that in Vertigo unconnected with visceral disease, and in persons under the age of fifty, there is not much danger to life, nor from what is most usually dreaded, viz. paralysis. Sudden and violent attacks of an intermittent character are unusually eccentric in origin, whereas a constant sense of uncertainty in movement, and a susceptibility to the induction of giddiness from the movement of passing objects, especially if combined with a cloudiness of intelligence, or rather a want of the usual clearness, indicates usually a centric disturbance. When, however, a severe attack occurs, without any palpable cause, to a person after the climacteric has been reached, a cautious prognosis must be given, and the more so if it be associated with vomiting, or constant nausea, tingling of extremities, the sense of pins and needles in one hand or foot, or of neuralgia of a group of muscles, or of those of one limb. The just fear in such a case is the fear of impending apoplexy. A discovery of dilated heart, of valvular disease of that organ, of degeneration of kidneys, with the presence of albumen in the urine, will make the prognosis more serious still. Organic disease apart, Vertigo has been known to exist during a long life, and indeed, unless some other suggestive symptoms are superadded, it cannot be considered a dangerous disease. In fact, the longer the complaint has existed in any given case, the less dangerous it appears to be.

ETIOLOGY.—The *direct proximate* cause of all Vertigo appears to be a disordered cerebral circulation; whereby, on the one hand, the special senses convey a false impression to the sensorium, or, on the other, a faulty co-ordination of muscular action is induced.

Of *remote causes*, it seems probable that any acute disease, or any sudden perversion of function of any important viscus on the body, may cause Vertigo, either directly or by reflex action. Thus we find stomachal vertigo as the commonest of all forms of the complaint, excepting only the invasion of all, or almost all, acute inflammatory diseases, the exanthemata, &c.; next, poisoning of blood, whether by disease, as from cachæmia, excessive smoking, intoxication or paludal poison; then organic disease of heart, of right or left side, after such disease has reached a certain point, which acts by altering the cerebral circulation in a twofold manner. In like manner the suppression of a long-accustomed hæmorrhage acts, whether it be in the form of epistaxis, bleeding from hæmorrhoids, or prolapsus ani, or from the menstrual flow ceasing too suddenly.

The rapid suppression of an extensive chronic cutaneous eruption is an acknowledged common cause, and it is explicable on the same principle.

VARIETIES.—The varieties of Vertigo may be practically divided into eccentric forms, or those arising from functional disorder of any viscus or viscera in the body; or centric, from organic disease in the brain itself, or by blood-poisoning. There is a third variety, important enough to demand separation from these groups. I have called it essential Vertigo. It is not associated with any other head symptoms, and there is no appearance of depraved general nutrition. It occurs mostly in persons about thirty years of age, and is a rare form of the disease, as compared with Vertigo arising from other demonstrable eccentric causes. In other respects, a patient suffering from it will declare himself in perfect health. In all the cases I have seen, the complaint has been associated with a decidedly weak heart, a feeble small pulse, and with symptoms I take to indicate a dilated right ventricle. Another characteristic may be said to be this, that it is not materially improved by remedies, unless these are accompanied by rest and freedom from anxiety of every kind.

STOMACHAL VERTIGO.—The most common and most tractable eccentric variety arises from disorder of stomach, or of functional derangement of the liver and upper part of the alimentary canal. It often occurs suddenly in the middle of the night, or without any warning at any period of the day, and in a state of apparent robust health. From its violence it suggests the idea of imminent danger. The following case may be taken as a type :—A merchant, some three hours after breakfast, after transacting some business of an exciting character, was quietly walking from a neighbouring office to his own, when he was suddenly seized with violent Vertigo. He reeled and immediately laid hold of an adjacent gas pillar ; he felt sick. Resting a few minutes, he felt the giddiness subsiding, and tried to walk ; but with the first step the Vertigo returned in greater violence, accompanied by a strange tightness of scalp. He asked a passer-by to assist him, and with the help of this second person managed, reeling or rolling, to reach his office, a distance of a few hundred yards. Seated in his chair, the symptoms gradually subsided; and in a few hours, after a free evacuation of the bowels, he was free from the Vertigo, but he felt weak and shaken, and complained of a heavy diffused headache. From a very careful examination these facts were elicited. The Vertigo seemed to be of both forms described at p. 175. He felt giddy in himself, and his legs were feeble, but the objects in the streets were also strange. The shop windows seemed moving forwards, passers-by were racing after each other, the ground felt to his feet uneven, billowy, as if elevated and depressed, and he felt constrained to lift his feet over the apparent elevations. Yet he was distinctly conscious of this illusion, and tried to conceal it. The headache occupied the entire head ; it was not acute ; it gave the sensation of weight rather than of pain ; it was not more frontal than vertical and occipital in its seat. There was no discoverable disorder of stomach or of any individual viscus, and, beyond the sudden attack of diarrhœa, nothing to suggest disorder of the abdominal viscera. He attributed the attack, and probably correctly, to having eaten very heartily a breakfast of which sausages and Devonshire cream formed a part, and to a hasty and very imperfect habit of mastication. During the ensuing month this patient had five separate attacks of the same violence, but without the same disturbance of bowels, and without being able to discover any cause, most assuredly not in the matter of diet, in which he had become exceedingly careful. Yet he was completely and permanently cured by the remedies adopted for stomachal Vertigo. As a matter of fact, it is very rare to find any positive signs of stomach disorder in these cases. They are named stomachal because remedies addressed to the stomach cure, and cure readily and quickly. With respect to the kind of Vertigo experienced, it does not, exclusively, take either of the two forms; it assumes both characters in some individuals. However, it is often so entirely connected with the appearance of external objects to the patient's eye, that the internal sense of giddiness is with difficulty made manifest. Curiously enough, it is rarely that patients complain of exaltation or defect of hearing, or of tinnitus aurium, although both these complaints are very common in the chronic stomachal forms of Vertigo. The *rationale* of the symptoms would appear to stand thus :—Digestion progresses satisfactorily up to a certain point, when, owing to some temporary cerebral excitement, perhaps of transacting business or of deep thought, the process is suspended, an irritation is conveyed to the blood-vessels of the brain, *viâ* the splanchnic or pneumogastric nerves, and a disorder of circulation and of brain nutrition follows, with a corresponding disorder of function of the particular parts of the brain affected. Like causes produce like effects ; and, moreover, in disorders of the nervous system it seems that a perversion of function, once induced, is easily re-induced, and by slighter causes. Hence it is not surprising that, if a patient has suffered from this acute stomachal form of Vertigo once, he will be subject to recurrent attacks.

Chronic stomachal Vertigo is of very common occurrence, and one often supposed to indicate the commencement of congestion, of organic disease of brain, of minute tissue change, premonitory of softening, or of threatening apoplexy ; and the treatment which has been adopted under such erroneous diagnosis has only served to render the Vertigo permanent. Patients in this form of disease do not usually complain of the common symptoms of dyspepsia. There is never any acute pain referred to the stomach after food; often there is a slight weight, a somewhat tender epigastrium, only however felt on deep pressure, evidences not so much of a perverted as of a slow digestion. Complaints are sometimes made of a pain radiating from the stomach to the back, to the cardiac region, or of a general undefined uneasiness about the entire epigastric region. Rarely can more than this be made out by a most careful cross-examination of the patient's stomachal symptoms. In the lower ranks of life, however, such as we find in hospital practice, we meet with all sorts of complications ; but that the symptoms appertaining to the stomach are not urgent may be inferred from the fact that patients do not seek advice for their relief, but for the Vertigo, and some steadily refused to admit there could be any disorder of the stomach, when remedies addressed to that organ afterwards cured the Vertigo. Additional symptoms of functional disease of other organs are, of course, in such a class of patients common, but they are found to be independent of, and to have little influence on, the Vertigo.

Very usual combinations of symptoms run thus:—Vertigo, pyrosis, leucorrhœa. Vertigo, menorrhagia, leucorrhœa, anorexia. Vertigo, weight of the entire head; relieved after food. Vertigo, vertical headache, nausea both before and after food. Vertigo, clavus, obstinate constipation, amenorrhœa. Vertigo progressive, weakness of sight, formicatio. Vertigo, tinnitus aurium, and partial deafness. In all these combinations the collateral diseases may be cured, and yet the Vertigo remains. There are several points of interest connected with chronic stomachal Vertigo which serve as a means of diagnosis from the graver forms of it. Thus it is never associated with loss of consciousness. There are intervals of hours in which the patient is perfectly free from it. It is made worse by excitement, by long fasting, and almost always the severe attacks occur when the stomach is empty. A stimulus in the form of wine or brandy relieves it; so also does food taken in small quantity. Closing the eyes to exclude objects in motion often relieves. During the attack a steady gaze on some fixed object mitigates the intensity of the sensation of giddiness. It is right to say that closing the eyes and the steady gaze are not invariably productive of relief, although subsequent treatment may prove Vertigo to have been stomachal.

In some cases the giddiness is slight, but almost constant; then it is usually associated with tinnitus aurium. More commonly it will occur several times daily, lasting from a few minutes to an hour, varying in degree, and accompanied by a singular general heaviness of the head, and a sense of heat at the vertex, which latter becomes aggravated when the Vertigo ceases. With respect to the peculiar form of Vertigo, no special conclusions serving the purpose of diagnosis can be drawn. Almost always unevenness of the ground is spoken of, or an illusive opening of the ground under the feet. Objects race in the eye of the beholder, and the patient feels going round with the objects he looks at when confined to a limited space, as in a small bedroom.

There are two varieties of chronic stomachal Vertigo which resist ordinary treatment. I allude to those forms complicated with, in some cases caused by, changes of tissue and alterations of the structure of the minute arteries, such as are known to occur in the persons of hard drinkers, or in those who have suffered from delirium tremens, and also in those who have suffered from latent or slight and irregular gout. It is in these cases we find the rarer forms of vertiginous perception,—as, for instance, when objects in the street or in the room appear turned upside down. In such persons vertiginous perceptions and movements last for days, and are often so severe as to confine the patient to bed, incapable of the slightest movement in the upright position without assistance. Nausea and disinclination for food are the only stomachal symptoms present.

Vertigo of the Aged is often stomachal, but equally often has no reference to that organ. As years are added, arteries become atheromatous, and otherwise diseased and obstructed, the circulation in the brain becomes irregular, we may have congestion in one place and anæmia in the other—a varying condition, abundantly sufficient to explain the frequent slight attacks of Vertigo in the aged. The essential condition of the brain is always one of anæmia. (Maclagan.) It is always to be remembered that the prognosis is always more or less unfavourable when Vertigo has commenced only in old age, on account of the known pathological condition of the nerve centres.

Essential Vertigo.—Some remarks have already been made on this variety. The following case will best illustrate its characteristics:—A gentleman, aged thirty-four, of considerable energy of character and great bodily vigour, has for three years suffered from almost constant, for the last two years quite constant, Vertigo. He is in comfortable circumstances, and has been very free from the ordinary anxieties of life. He has led a temperate country life, and has never had syphilis, gout, or rheumatism. Excepting the Vertigo, he has enjoyed excellent health. He says the giddiness came on gradually, and was at the commencement so slight that he can hardly fix the time of its first appearance. In kind it was subjective. At first, he found himself giddy on dressing in the morning; he felt as if he had taken too much wine overnight, and his legs were weak, and his gait unsteady. After breakfast he was well. The attacks became more prolonged, and occurred at various periods in the day; and now he is rarely free from a sense of uncertainty rather than positive giddiness. Occasionally he becomes worse, and is obliged to sit down to prevent falling. He has no confusion or muddiness of intellect, has never lost consciousness, has no complaint of headache, dyspepsia, or disorder of any other kind. After many examinations, I have not been able to discover even functional disorder in any of the abdominal viscera. He has a soft, small, compressible pulse. The impulse of the heart is not visible; the area of dulness enlarged laterally to the right; the sounds are feeble, close to the ear, and too clearly audible to the right of the sternum. This patient has undergone a variety of medical treatment in the hands of various practitioners, including strychnine, which was pushed to the verge of producing involuntary spasms in the limbs. He has tried the hydropathic treatment, has passed a season at Vichy, but has not been able to find the slightest benefit. Very careful diet has not altered his condition, but excesses of any kind make him worse. Although there is no evidence of valvular disease, yet I cannot help connecting the feeble heart, and perhaps enlarged right ventricle, with a disordered cerebral circulation, which is itself the proximate cause of the Vertigo. I have met with two cases of this kind which were apparently hereditary. The father of one of them is now seventy-one years of age; he suffers from spasmodic asthma, and has been the victim of Vertigo for the past thirty-five years.

Vertigo from Overwork ranks next in frequency to the stomachal variety. It occurs in young persons who are underfed as well as overworked, as in some sempstresses; in the middle-aged, who to spare diet add various irregularities, as well as in the temperate and well-fed, who are constantly subject to mental anxiety and excitement. The attacks of Vertigo are of short duration, occur at intervals of some hours or days, after prolonged exertion, or poorer diet than usual; it is only a sense of the abnormal appearance of external objects at first, and occurs only on movement; it becomes more frequent, and then assumes, in addition, the character of an internal feeling of dizziness; the recumbent position always relieves, but does not even temporarily cure it. It is often complicated with stomach disorder, as anorexia, rarely nausea, with constipation, and in the female sex with menstrual irregularities. But the simple stomachic remedies do not remove, they scarcely mitigate, the Vertigo. Patients complain of a want of clearness of intellect, an incapability of sustained mental exertion, together with occipital heaviness or headache. In the worst cases, irritability of temper, restlessness, a sense of impending evil, and more rarely insomnia, are added. Sometimes the Vertigo is so easily induced by the appearance of objects in motion, that the patients are unable to go into the streets. In such cases there are functional ailments of other organs, palpitations, and lumbar pains, accompanied by the passing of phosphates in the urine. Oxaluria is not an unfrequent complication. Indeed, there is a general lowering of vitality, a universal deprivation of nutrition, and corresponding diminution of power, of which the Vertigo is only one of the exponent symptoms. This is the form of Vertigo which most often amongst business men precedes softening of the brain. Vertigo from irritation of the auditory nerve has been noticed by my late colleague, Dr. Brown-Séquard. He mentions, in his Physiology of the Nervous System (p. 195), this result produced by injecting cold water into the ear, and also by the topical application of nitrate of silver; and he suggests that such applications act in a reflex manner on the blood-vessels, producing temporary anæmia and a disordered circulation and nutrition of the brain, resulting in the production of Vertigo.

Ménière, in 1841,[1] established before the Academy of Medicine in Paris, that certain affections of the ear produced a series of symptoms closely resembling those attending disease of the brain, as Vertigo, dulness, uncertain walk, occasional circus movement, and even falling down, accompanied also by nausea, vomiting, and syncope. He gives also a case of a young girl who, travelling one cold night in winter during menstruation, was seized with sudden and complete deafness. Her chief symptoms were continual giddiness and irrepressible vomiting, produced by the slightest movement. She died on the fifteenth day; yet no trace of disease was to be found in the brain, cerebellum, or spinal cord. The semicircular canals only exhibited traces of disease; they were filled with reddish plastic lymph. Other cases are on record, by French authors, of a similar kind, some having associated with the Vertigo dysæsthesia; that is to say, the slightest noise producing positive and even severe pain in the affected and deaf ear. It is remarkable that such cases may terminate fatally, without presenting one single symptom of feverish reaction, and without any extension of disease to the brain (Trousseau). It is well known that Vertigo in animals may be produced by puncture of the semicircular canals, as well as by wounding various parts of the base of the brain. Further information will be found in Dr. Brown-Séquard's Physiology of the Nervous System, and in the works of Schiff, Flourens, Magendie, and Claude Bernard. These are, however, matters rather of physiological than medical interest. Cases of vertiginous movements arising from disease of brain are common, such as a tendency to gyrate, to fall forwards, to one side, or backwards; but we are not able in the present state of science to draw accurate conclusions as to the seat or nature of the disease, unless it be one of a group of symptoms involving paralysis, or having other special marks of disease in a particular locality. Vertigo accompanies, to a greater or less degree, almost every organic disease of the brain, and every acute affection of this organ. Its value as a sign of disease clearly depends on its association with other symptoms; and it can only be properly appreciated in connexion with a study of those diseases of which it forms a minor part.

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[1] Bulletin de l'Académie de Médecine, vol. xxvi. p. 241; Gazette Médicale, 1861, vol. xvi. pp. 88, 239, 597.

In a large number of hospital cases there is the association of Vertigo on movement with tinnitus aurium and partial deafness. The tinnitus and deafness appear first, and the Vertigo follows. I have never been able to trace anything like suddenness in the invasion of these symptoms; their accession is always gradual, and unassociated with pain in or about the ear, or with symptoms of fever. Persons in fair average health, and without any stomach or other obvious disorder, suffer most. There seems to be some mischief of a very slow kind going on, perhaps in the semicircular canals connected with the circulation, analogous to the more acute cases recorded by Ménière. The occasional value of counter-irritation, and of iodide of potassium and of small doses of mercury, confirms this view.

TREATMENT.—Stomachal Vertigo, in its acute and chronic forms, often yields to a very simple method of treatment. This consists in the exhibition of alkaline remedies and of alterative aperients continued steadily for some weeks, to be followed by bitters, and especially by the use of nux vomica or strychnia. The alkaline treatment is to be used after meals so as to neutralize any formation of acid, and to excite a freer secretion of gastric juice; the tonics to enable the stomach and bowels below to perform completely their functions. Stomachal vertigo of the severest kind yields most readily to the influence of these remedies. At the same time food is to be taken in small quantity, to be carefully masticated, at regular periods; and, for drink, Vichy water mixed with a small quantity of brandy acts most efficiently. All kinds of malt liquors are to be forbidden, whilst general hygienic measures are to be adopted. The splash bath in the morning, early retiring to rest, sleeping on a mattress in a large airy bedroom, are great adjuvants to the treatment. Freedom from the cares and anxieties of business are not less necessary. In all varieties of Vertigo it is wise to commence the treatment as if the case were stomachal, not simply because the case may turn out one of this variety, but because stomachal disorder may complicate any variety of the malady. The chronic forms of the complaint are more difficult of cure, but the same principles apply, and the treatment must be varied according to the peculiarities of the individual case, always remembering, however, that it will be wise to attack and remove the complications which are associated with it, before making a special treatment of the Vertigo. In more obstinate forms of disease connected with tissue degeneration, intemperance, or with chronic gout, measures adapted to these several conditions will of themselves relieve the Vertigo, and prepare the way for the restoration of tone and improvement of nutrition, on which any hope of a great amelioration or cure must depend.

The Vertigo of the aged demands wine, and any plan of treatment which the case may demand must be associated with stimulants, unless (a very rare occurrence) the Vertigo be premonitory of meningitis, and is accompanied by heat of the scalp and some congestion of the conjunctiva. A most effective combination for Vertigo of the aged consists in very small doses of the bichloride of mercury, with tincture of iron and cantharides. In Vertigo from overwork, in the well-fed there are usually present restlessness, insomnia, depression of spirits, and a vague feeling of unhappiness or impending evil, for the relief of which I have found great help in bromide of ammonium, given in an effervescing form, with the addition of cascarilla. Amongst the poor, where scanty food accompanies overwork, this remedy is not of such value ; we shall gain more from measures calculated directly to improve nutrition, and from slight stimulants frequently repeated. Brandy or wine, under these conditions, is a better tonic for a time than bark or quinine, which will be found most appropriate afterwards. The solutions of the hypophosphites are also especially valuable.

Essential Vertigo is most benefited by a long course of citrate of iron and strychnia, given in an effervescing form, alternating month by month with tincture of larch and small doses of digitalis. The local application of belladonna does good, although there may be neither pain nor palpitation to suggest its use. I believe it is a direct tonic to the muscular tissue of the heart, in which respect it resembles the preparations of larch, and perhaps also of digitalis. The usual conditions of rest, freedom from care and anxiety, are, of course, as essential as in the other varieties of the disease. The treatment of Vertigo arising from grave disease of brain, from softening of its structure, from aneurism, or tumour, must be involved in the treatment of these diseases.

CHOREA.

By C. B. Radcliffe, M.D., F.R.C.P.

Chorea is the disease known as St. Vitus's dance in this country, as the dance of St. Guy in France, and as the dance of St. Weit in Germany, St. Guy being the name which is the French equivalent of St. Vitus or St. Weit. It is chiefly characterised by irregular clonic movements of the voluntary muscles, and by weakness more or less approaching to paralysis in the same parts.

1. Symptoms.—Chorea is sketched for the first time in the writings of the English father of medicine. "St. Vitus's dance," says Sydenham, "is a sort of convulsion which attacks boys and girls from the tenth year until the time they have done growing. At first it shows itself by a halting, or rather an unsteady movement of one of the legs, which the patient *drags*. Then it is seen in the hand of the same side. The patient cannot keep it a moment in the same place; whether he lay it upon his breast or any other part of the body, do what he may, it will be jerked elsewhere convulsively. If any vessel filled with drink be put into his hand, before it reaches his mouth he will exhibit a thousand gesticulations, like a mountebank. He holds the cup out straight, as if to move it to his mouth, but has his hand carried elsewhere by sudden jerks. Then, perhaps, he contrives to bring it to his mouth; and if so, he will drink the liquid off at a gulp, just as if he were trying to amuse the spectators by his antics."

The symptoms of the fully developed disorder, as the following case will serve to show, are marked enough and characteristic enough.

Case.—Mary C——, aged 11, admitted into the Westminster Hospital, under the care of the writer, on the 12th of March, 1864.

She is suffering from pains in the limbs, slight feverishness, and some tenderness and fulness in the right wrist, the pains in the limbs being chiefly in the right arm. The day before, she got drenched to the skin in a shower, and was obliged to remain in her wet clothes for some time. She is a bright-faced, good-looking, exceedingly pale child, the reverse of dull and stupid in every way, never strong, but never ill, except with severe convulsions when cutting her first teeth. Her mother had four or five epileptic fits about the time of puberty.

March 14.—The pains in the limbs are better; but the tenderness and swelling of the wrist has somewhat increased in the right wrist, and extended to both the ankles. The pulse is 100, and slightly irregular; the action of the heart is a little excited, and there is a slight systolic bruit at the apex. The skin is moist, and the perspiration has a sourish smell. The appetite has gone altogether, and there is some thirst. The medicine ordered contains iodide of potassium and bicarbonate of potass.

March 20.—The fulness and tenderness of the joints have disappeared, and so have the thirst and want of appetite; but the cardiac murmur is more, rather than less, marked. Ordered to get up, and to have cod-liver oil.

March 27.—She has just been greatly frightened by seeing a patient close by die suddenly, and is now crying and sobbing bitterly. Previously to this she had been playing with another child in the ward, and was to all appearance quite well.

March 29.—A marked change has taken place since the last visit. There is now great restlessness, and impatience, and fretfulness, with curious wriggling, fidgety movements in the right arm. Her sleep has been much disturbed, and twice in the night she got up and went to the sister, crying and saying she was frightened. Four ounces of wine were ordered.

March 30.—The restlessness is much increased; but, instead of impatience and fretfulness, there is now evident dulness and listlessness. The right arm is continually jerking about, and in attempting to walk the right leg both jerks and drags. Though right-handed, the left hand is used in feeding, and on inquiry it is found that the right hand is useless for this purpose. The speech is thick. All the joints feel strangely loose. There is some difficulty in swallowing, and the food is rolled about in the mouth some time before disposing of it in the usual way. The features twitch and twist a little, but not much. The tongue is put out and kept out without difficulty, and it is not particularly unsteady. All the disordered movements are much increased by trying to be still, and during fits of crying and fretting, which fits are not uncommon. The pulse is quick and small, the hands are cold and rather damp, and there is a constant wish to huddle over the fire; the bowels are very sluggish; the urine is neutral, and readily becomes offensive. Ordered to have hypophosphite of soda and cod-liver oil thrice daily, and a single dose of castor oil.

March 31.—The restlessness is much increased, and the tossings and jerkings have become almost general. Standing and walking are barely possible, partly from the jerks and tossings of the limbs, but chiefly from the weakness of the right leg. The grasp of the right hand is also much weaker than that of the left. The right foot is a little more sensitive to pinching than the left. The features are almost continually being twisted into the oddest grimaces, but when at rest they are so wanting in expression as to give the idea of extreme silliness. Indeed, the expression is so changed as to make it difficult to identify the patient as the bright-faced, intelligent girl she was when admitted into the hospital. The speech is quite inarticulate, her only question or answer being "um," with a snort. Saliva dribbles from the mouth, and even food, which she can only now get by being fed, is scarcely kept from falling out of the mouth. Mastication and swallowing are both matters of much difficulty. The tongue is unsteady, but it can be put out and kept out by an effort. The pupils are dilated and sluggish—the left especially. The pulse is quick and weak, but not irregular; the hands are cold and moist. The movements are suspended during sleep, but sleep itself is only in comparatively short catches. The same medicines to be continued, with brandy and milk at short intervals, in addition four ounces of brandy being given in the twenty-four hours.

April 5.—No very material improvement. The grasp of the right hand a little stronger perhaps, and the right leg dragging and jerking not quite so much.

April 14.—A marked improvement. The gait is much more firm and steady; the features are less vacant and less discomposed; the sleep is comparatively sound; the appetite is better; the hands are warmer; the pupils are now fairly sensitive and equal in size, but the speech remains inarticulate, and the child has still to be fed. No change in the treatment.

April 21.—The irregular movements of the arms and legs are nearly at an end; the features are comparatively at rest, and the expression of intelligence has returned; the speech is distinct now, but the voice is low, and the articulation slow; the gait is slouching, but there is no dragging in the right leg; the power of self-feeding has eturned, though the left hand is still made use of rather than the right, and the sleep is sound and refreshing. No change in the treatment.

May 1.—Nearly well.

May 14.—Discharged well, except that there is still a systolic bruit at the apex of the heart.

In this case the salient points are, the age and sex, the movements, the paralysis, the numbness, the dulness and listlessness, the relations to rheumatism and heart-disease, the absence of fever, and the neutral urine; and the noticing of each of these points in turn will serve to bring out the general features of chorea in its ordinary form.

Age and Sex.—Sydenham states that chorea, for the most part, attacks children between the tenth and fourteenth year of their age, who have not reached the time of puberty. Sir Thomas Watson considers these limits to be too narrow, and extends them to the period of the second dentition on the one hand, and to that of puberty on the other; nay, he extends them still wider, for he states that now and then, but only exceptionally, cases occur as early as 4 or 5, and as late as 20 or 25 years of age. Up to nine years of age the two sexes appear to be equally liable; after this age females become much more liable than males, in the proportion of nearly 5 to 2.

Of 422 cases treated as out-patients at the Children's Hospital in Great Ormond Street, and tabulated by Dr. Hillier, the numbers of each sex at different ages were as follow:—

	Males.	Females.	Total.
From 3 to 6 months	1	2	3
,, 6 ,, 12 ,,	1	4	5
,, 12 ,, 18 ,,	1	1	2
,, 18 ,, 24 ,,	1	3	4
,, 2 years to 3 years	3	3	6
,, 3 ,, 4 ,,	6	5	11
,, 4 ,, 5 ,,	4	16	20
,, 5 ,, 6 ,,	7	23	30
,, 6 ,, 7 ,,	18	30	48
,, 7 ,, 8 ,,	17	34	51
,, 8 ,, 9 ,,	17	41	58
,, 9 ,, 10 ,,	23	57	80
,, 10 ,, 12 ,,	23	81	104
Total	122	300	422

At the Children's Hospital patients are not admitted above the age of 12, so that these statistics do not include cases from 12 to 15 years of age,—that is, about the age of puberty; but judging from other statistics, as Dr. Hillier says, "it does not appear that the period of puberty is more prone to the disease than the period between the second dentition and puberty." Thus, of 100 cases occurring at all ages, and tabulated by the late Dr. Hughes in the Guy's Hospital Reports, 29 were between 12 and 15 years of age, 9 being males and 20 females; at 15 years of age there were 5 females and 1 male. After puberty, chorea is comparatively rare. In 96 cases, of which the statistics are given by Dr. Ogle, 19 were above 15,—the ages being 2 at 16, 6 at 17, 2 at 18, 1 at 19, 2 at 20, 2 at 21, 1 at 23, 1 at 24, 1 at 26, and 1 at 43; and of these 19, 16 (including the one at 43) were females. And of 17 cases of chorea during pregnancy which proved fatal, which cases form the basis of an excellent paper by Dr. Barnes on chorea in pregnancy, the ages range between 17 and 24, with one exception, in which the age was 47.

The Movements.—These are the most characteristic feature of chorea. They are clonic spasms, unattended by pain, and, as Dr. Hillier says, "something like the restless movements of a child put out of temper." Usually they are more marked on one side of the body than on the other, and at first they may be confined to one side. Not unfrequently they make their appearance first in one arm, then in the leg of the same side, then in the face, then in the arm and leg of the other side. They are always increased by any attempt to exercise the will, or under any emotional excitement; and they are, for the most part, put a stop to by sleep. As a rule, it is enough to see the movements to recognise at once the nature of the disorder; and some of the movements of the face are very characteristic. Still it does not do always to reckon upon finding movements which are looked upon as characteristic. Thus, in the case which has been cited as a text, the tongue was not put out with a sudden jerk after a pause, and retracted with equal suddenness, after the manner which is described as specially characteristic of chorea; and most certainly this case is by no means exceptional in this respect.

Paralysis.—Want of muscular power is shown by the readiness with which the patients become tired, and by the slowness with which they recover from fatigue, as well as in the soft, flaccid, and wasted condition of the muscles when the disease has continued for some time. But this is not all which may be noticed, especially in those patients in which the choreic movements are confined to one side; for, in these cases, there is usually a want of power in the affected muscles which must certainly be spoken of as a slight degree of paralysis. This want, as Sydenham said, "often shows itself by a halting, or rather unsteady movement of one of the legs, which the patient *drags*:" or, still more frequently, it may show itself in the helpless way in which the arm almost immediately *falls* when it is held out, for it is easy to see that this falling is a phenomenon which has much more to do with paralysis than with choreic movement.

As positive evidences of paralysis, must be reckoned the loss of speech, the loss of facial expression, the loss of the power of swallowing, the inability to use the hands for the purposes of feeding, the looseness of the joints, all of which symptoms were present in a marked degree in the case which has been given, and one or other or all of which, in a greater or less degree, are usually present in all cases. Paralysis, indeed, is seen to be a marked feature in chorea, if only the attention is not allowed to be entirely absorbed by the contemplation of the movements; and in some extreme cases it may be so marked as to lead to incontinence or retention of urine, or to involuntary stools. Usually, also, the muscles which are most affected by the movements are those which are most paralysed. Nor is the connexion of chorea and paralysis altogether out of order; for in many cases of paralysis properly so called, the paralysed parts are affected by movements which, without question, are not remotely akin to those of chorea.

Numbness.—This symptom is sufficiently marked in many cases to be detected without difficulty, especially in the parts in which the movements are most marked, but it is never as prominent a symptom as in hysteria; and the same remark applies to the opposite condition of over-sensitiveness. Trousseau says that numbness, when present, is usually accompanied by tingling.

Dulness and Listlessness.—The vacancy of the features resulting from the semi-paralysed condition of the features, which may be so extreme as to suggest the idea of idiotcy, must not be taken as the gauge by which to measure the mental condition of the patient. Matters mentally are certainly not so bad as they look; still there is always more or less dulness and listlessness—dulness and listlessness rather than fretfulness, and undue excitability, as in the ordinary hysterical condition. It is to be remembered, also, that the children attacked by chorea are commonly distinguished by vivacity and restlessness of disposition.

Rheumatism and Cardiac Disease.—In his excellent digest of 300 cases of chorea, occurring in Guy's Hospital, the late Dr. Hughes ascertained that, "out of 104 cases in which special inquiries were made respecting rheumatic and heart affections, there were only 15 in which the patients were both free from cardiac murmur, and had not suffered from a previous attack of rheumatism." Nor is it possible to get over this fact by imagining that the pains of the supposed rheumatism may have been simply neuralgic, and the cardiac murmur merely anæmic, for in 11 out of 14 cases of death from chorea recorded in this paper, there were actual vegetations upon the cardiac valves. Dr. Romberg says, "The rheumatic predisposition, noted by English medical men, was rarely traceable in the cases presenting themselves to my observation;" but this opinion is not that of other German writers. Dr. West, who once had doubts as to the frequency of the connexion between rheumatism and chorea, now believes that the rheumatic diathesis is a powerful predisposing cause of chorea. M. Rogers says, "The coincidence of chorea and rheumatism is so common a fact, that it ought to be regarded as a pathological law, just as much as the coincidence of heart disease and rheumatism:" and again, "The child affected with rheumatism is, after a longer or shorter interval, threatened with chorea; and the child affected with chorea is sooner or later menaced with rheumatism." It may also, as Dr. Tuckwell points out, explain why it is that in adults rheumatism and chorea do not go together as they do in earlier life, that in earlier life rheumatism is far more frequently complicated with heart-disease. "The younger the patient," as Dr. Hillier remarks, "the more frequently is rheumatism accompanied by endocarditis." Cardiac disease is also very common in chorea. Thus, in 37 cases of which notes were taken by Dr. Hillier, there was probably organic disease of the heart in 25, and of functional derangement in 4, whilst in 8 only was there no sign of cardiac disturbance. Heart disease, however, does not necessarily point to rheumatism in children. On the contrary, it may follow scarlet fever or measles, and in some cases it may come on without any very obvious reason. But, be the cause of the heart disease what it may, heart disease is a common accompaniment of chorea, in the refractory cases especially.

Absence of Fever.—Fever does not figure among the necessary symptoms of chorea. In fact, chorea is essentially a feverless malady. Not unfrequently, also, there are signs which point to a condition of circulation the very opposite to that which is met with in fever, such as coldness and clamminess of the hands, a disposition to chilblains if the weather be at all cold, pastiness or puffiness of certain parts of the skin, anæmic vascular murmurs, and the rest. In some instances, it is true, the temperature is increased; but such increase according to my experience, is only met with in mixed cases of chorea, where delirium is a marked feature, and where the movements point to delirium rather than to chorea, and then only exceptionally, and therefore it may have nothing to do with the chorea. Moreover, increase of temperature is not always a sign of fever in the ordinary sense of the word, for it is a fact, not unfrequently verified, that the temperature often rises remarkably in the moribund state, and that for some time after death the corpse may give a disagreeably *hot* sensation to the touch. Nor is an argument to the contrary to be found in the relation of chorea to rheumatism. Chorea may occur before or after rheumatic fever, but not along with rheumatic fever. This is the plain fact. Indeed, the very connexion of chorea with rheumatism, when properly understood, may be only one other proof that chorea is associated with a state of wanting vigour and activity in the circulation, for most assuredly a weak circulation and a lymphatic habit generally is the state of things which is likely to be present in persons who are prone to rheumatic fever. Moreover, it not unfrequently happens that the symptoms of chorea are suspended by the accidental development of scarlet fever or some other febrile disorder, and that they return again when the state of feverishness passes off.

Neutral Urine.—In the case which serves as my text the urine was neutral, and readily becoming offensive; and so far as my experience goes, this is the case generally. In some cases, however, the urine seems to be of unusually high specific gravity, as has been shown, first of all by Dr. Walshe, and afterwards by the late Dr. Todd and by Dr. Bence Jones. In a case of acute chorea, of which Dr. Walshe gives the history in detail, lithates were deposited in large quantities during the first few days, then urea was found to be present in great excess, then oxalates made their appearance, and last of all there was a copious precipitation of oxalates; and in another case, given in the Clinical Lectures of Dr. Todd, the specific gravity of the urine was never below 1·019, and often as high as 1·030 or even 1·035, and as a rule urea and oxalate of lime, but especially lithates of ammonia, were present in considerable excess. The state of the urine requires to be more carefully inquired into. In two cases I found, for a short time only, some excess of urea, and a thick deposit of lithates on cooling; but this state of things soon changed, and what I noticed chiefly was the rapidity with which the urine lost its acidity, and threw down phosphates.

The case of chorea which has served as the text for these comments is a little more marked in its symptoms than the average of cases. Usually, indeed, the speech is thick and confused, not lost, and the use of the hands is not so completely taken away. Usually, also, paralysis is a less prominent phenomenon. The symptoms are, in fact, infinitely varied; and as they are toned down on the one hand, or exaggerated on the other, chorea may be a most trifling disorder or a very grave malady. In its most trifling form chorea may be nothing more than a grimace, or a shrug of the shoulders, or a catch in the speech, or some other odd or awkward involuntary movement, which in many instances appears to be little more, or no more, than an unchecked bad habit. In its gravest form, on the contrary, few diseases are more distressing to witness—the patient tossing ceaselessly to and fro, unable to walk or even stand, turning, writhing, dashing about, and only kept in bed by being strapped down or fenced in; without speech, perhaps with the lips torn, chapped, and bleeding, by being, in spite of all we can do to prevent it, continually drawn into the mouth and munched between the teeth, which themselves, in some instances, are actually ground down and even forced from their sockets; with the elbows and hips and other prominent points made raw by constant rubbing against the bedding—a sight which is forced upon one, for no care can keep the bedclothes in their place—sometimes raving, and never sleeping, until death comes to the rescue. Once seen, indeed, it is not easy to forget a scene so sad as that presented by chorea in its gravest form, a scene than which there is none sadder in the whole range of diseases, hydrophobia itself not excepted.

2. EXCEPTIONAL FORMS OF CHOREA.—Allied more or less closely to chorea in its severest form is a disease which was first described by Dr. Dubini, of Milan, about twenty years ago, under the name of *electric chorea.* This disease seems to be peculiar to certain districts of Lombardy. Its symptoms are:—(1) Certain choreic or convulsive shocks in the limbs, repeated with a certain regularity of rhythm, persisting with scarcely any intermission for days, or even weeks, and followed by paralysis, and, it may be, atrophy of the affected parts; (2) certain tonic convulsions of great violence, affecting the muscles in which the choreic convulsions are manifested, and occurring in not unfrequent paroxysms; (3) epileptiform attacks, sometimes general, sometimes partial; (4) certain head-symptoms, such as cephalalgia, delirium, and coma. One or other of these groups of symptoms may be predominant in different cases. Electric chorea may be either acute or chronic, and in either case its termination is almost always in death. As a rule, it begins quietly, and is in no great haste to assume its serious characters. As a rule also, a delirium, lasting for some days and ending in coma, ushers in the fatal termination; but not unfrequently death is brought about more speedily and suddenly in an epileptic paroxysm. The electric shocks which form so conspicuous a feature in the disorder, occur very frequently, as often as thirty, sixty, or a hundred times in the minute, and they are often, if not always, accompanied by feelings of pain, tingling, or cramp in the same parts, by vertigo, and by humming or singing sounds in the ears. At first the digestive organs are but little affected, but after a time the appetite fails, and gastralgia and frequent vomiting add to the distress. Fever, indeed, is not seldom present. The mean duration of the disease is from forty to seventy

days, if we except a few acute cases in which death happened in a few days with urgent cerebral symptoms.

Electric chorea was the name chosen for this disease by Dr. Dubini; *typhus convulsivo-cerebralis* was the name selected by Dr. Frua, a colleague of Dr. Dubini's in the same great hospital in Milan, who saw many cases, and whose description of the disease immediately followed that of this last-named physician; and *myelitis convulsiva* was the name made use of by Dr. Hörtel, in his account of the disorder. This difference of nomenclature shows how differently various observers were struck by what they saw, and proves, at the same time, what is plain from their description, that electric chorea has not, perhaps, the strictest claim to be admitted into the category of choreic affections.

St. Vitus's dance, however, is the very Proteus of diseases, and many strange maladies have to be passed in review before the description of all its various forms is complete.

The disease to which the name of *St. Vitus's dance* was originally given was of an epidemic character. It broke out at Strasburg in 1418, close upon the heels of the black death. It was, in fact, a fresh outbreak of a dancing epidemic called the *dance of St. John*, which made its appearance at Aix-la-Chapelle in the summer of 1374, and then spread like wildfire over the whole of Germany and the countries to the north-west. This dance of St. John appears to have been characterised chiefly by paroxysms of extravagant dancing and leaping and howling and screaming. In some cases the head was filled with ecstatic visions in which St. John was a prominent object; in others, the most frantic excitement was produced by certain sights or sounds. Sometimes the dancing movements were ushered in by symptoms of an epileptiform character: usually they were accompanied and followed by the most distressing flatulency: almost always they were carried on until they came to an end from sheer want of strength. For nearly two hundred years society was disorganized by persons suffering from this demoniacal disorder, and by rogues who simulated it for sinister purposes. Dr. Hecker tells us that the feast of St. John the Baptist was always held as a day of wild revelry; and that at the time when this strange malady made its appearance, the Germans were in the habit of mixing up with this Christian ceremonial an ancient pagan usage—the kindling of the "nodfyr." It was the custom on these occasions to leap through the flames of this fire, and t
consider that a year's immunity from the disease was gained in thi
way; and in this leaping run mad, Dr. Hecker thinks, we have th
origin of the dance of St. John.

In its main characteristics the dance of St. Vitus does not appea
to have differed from the dance of St. John. The difference of nam
was owing to this—that at the first appearance of the disease i
Strasburg, the sufferers, real or pretended, were so numerous that th
city authorities divided them into companies, and appointed person
whose duty it was to conduct them to the chapels of St. Vitus nea

Zabern and Rotenstein, as well as to protect and restrain them by the way. They were taken to these chapels in consequence of a legend, invented conveniently for the occasion, which represented that this St. Vitus, when suffering martyrdom under Diocletian, A.D. 303, had, in answer to prayer, received power to protect from the dancing mania all those who observed the day of his commemoration, and fasted upon its eve. At any rate, to the shrine of St. Vitus these people went, and there priests were ready to sing masses, and to perform other services fitted for the occasion; and thus the name of the disorder became changed from the dance of St. John into the dance of St. Vitus.

Attention was first prominently directed to these two dances, at the times which have been mentioned, but there is good reason to believe that they had been known a long time previously.

At the beginning of the sixteenth century, a change had taken place by which these disorders had become less unlike disorders which are now classed under the head of chorea. This is evident from the description given by Paracelsus and other competent observers. At this time these maladies were characterised by frequent fits of hysterical laughing or crying, by odd movements, and now and then by fits of dancing, but not by the howling or screaming or mental delusions or distressing flatulency of former days. In some instances, also, the propensity to dance was not irresistible. Still, now and then the disorders in question appeared in their old form, and Dr. Hecker tells us that so late as 1623 some women were in the habit of paying a yearly visit to the chapel of St. Vitus, in the territory of Ulm, in order that a dance at the altar there might save them from dancing elsewhere against their will, until the same time next year.

Almost contemporaneously with the dance of St. Vitus, a dancing malady, called *tarantism*, appeared at Apuleia, and spread from thence with great rapidity over the rest of Italy. This malady was attributed to the bite of a tarantula, or ground-spider, common in the country; but it is more probable that undue fears as to the evil consequences of the bite—fears arising easily in the gloomy and despondent temper of the times—had more to do in causing the malady than the bite itself. Those who were bitten remained dejected and stupified, or else, becoming greatly excited, went about laughing, singing, or dancing. In any case, they were utterly unable to restrain themselves if acted upon by music of a certain kind. A bacchantic furor was excited by the first notes, and as the performance went on hey would dance, and leap, and shout, and scream, until they fell down from sheer exhaustion. Some colours appeared to have excited hem, others to have calmed them. Some had a strong disposition o rush into the sea; many were carried away by strong sensual passions into deplorable excesses. Some, again, were tormented by he flatulent distress which was a symptom in the dance of St. John. n this malady, music was looked upon as the only remedy, and the country everywhere resounded with the merry notes of the tarantella. The favourite instruments were the shepherd's pipe and the Turkish drum. It was supposed that the poison of the tarantula was diffused over the system by the exercise of the dancing, and expelled along with the perspiration. It was customary for numerous bands of musicians to traverse the length and breadth of the land during the summer months, and the seasons of dancing at the different places were called "the women's little carnival," "carnavaletto delle donne," for it was the women, more especially, who conducted the arrangements, and defrayed the expenses. Tarantism continued in Italy long after the dance of St. Vitus had died out in Germany; indeed, the epidemic can scarcely be said to have been at its height until the middle of the sixteenth century.

It would seem also that the *tigrétier* or dancing mania of Abyssinia, a malady occurring most frequently in the Tigré country, is, in some respects, not unlike the ancient dances of St. Vitus and St. John. Beginning with violent fever, this malady soon turns to a lingering sickness, in which the patient becomes reduced to the last degree of emaciation and exhaustion. This sickness may continue for months, and end in death if the proper cure be not sought after. The first cure, which is also the cheapest, is one in which a priest ministers. It is a kind of water cure, with a blessing superadded. If this fail, the aid of music is appealed to, and arrangements are made for a prolonged performance. The place chosen generally is the market-place. Under the influence of the music the patient soon bestirs herself, and begins to leap and dance in the maddest manner possible, and, having begun, she goes on in the same way until the day is nearly, and the musicians altogether, spent, and then she starts off, and runs until her legs refuse to carry her any further. Then a young man who has followed her fires a gun over her head, and, striking her on the back with the flat of a broad knife, asks her name, when, if cured (she had never uttered this name during her strange illness), she repeats her Christian name. After this she is re-baptized, and considered convalescent. The account of this extraordinary affection is by Mr. Nathaniel Pearce, who lived nine years in Abyssinia, who saw what he describes, and who published th story about thirty years ago.

A place in this strange category of disorders must also be concede to those extravagant leapings and dancings which have been met wit at various times among certain sects of religious enthusiasts—th jumpers of this country and America, the "convulsionnaires" in Franc and the victims of "leaping ague," who some time ago startled an shocked the grave people of Scotland. These latter enthusiasts complained of pains in the head and elsewhere, and soon afterwards the began to suffer at certain periods from fits of convulsion and fits dancing. At these times they acted in the maddest way, distortin their bodies, springing to a surprising height, or running with amazin velocity until they fell down exhausted. When confined in cottag a favourite practice was to leap up and swing about among the beams supporting the roof. The effects of music do not appear to have been tested.

The time for a general visitation of maladies such as these, would appear to have passed by, at least, in this country; but there are still to be met with, now and then, isolated cases which have some claim to be included in the same category—cases distinguished by involuntary leaping, turning, or rushing backwards, forwards, or sideways. One of these, often quoted before, is recorded by Mr. Kinder Wood; and this, with two which have fallen under my own observation, may serve as illustrations.

Mr. Kinder Wood's patient was a young married woman who had suffered for some time from headache, nausea, quick involuntary movement of her eyelids, and various contortions of the limbs and trunk. The paroxysms themselves were not always of the same kind. At one time she would be violently and rapidly hurled from side to side of the chair in which she might happen to be sitting, or else, suddenly gaining her feet, she would go on jumping or stamping for a while, or she would rush round and round the room and rap with her hand each article of furniture that lay in her course. Or she would spring aloft many times in succession, and strike the ceiling with the palm of her hand, so that it became necessary to remove some nails and hooks which had done her an injury. Or she would dance upon one leg, with the foot of the other leg in her hand. These movements always began in the fingers, and the legs were not affected until the arms and trunk had been first seized upon. Noticing a rhythmical order in some of her movements, as if they were obedient to the memory of some tune, a drum and fife were procured, and the result of playing upon these instruments was, that she immediately danced up to the musicians as closely as she could get, and continued dancing until, missing the step, she suddenly came to a standstill. On another occasion a continuous roll of the drum at once put a stop to the dancing movements. Afterwards, the drum was used in this manner with the happiest results, and at the end of a week these movements may fairly be said to have been stopped and cured in this way. Unfortunately, however, the drum and the fife were alike found to have lost the power on two subsequent occasions when the dancing recurred. These strange paroxysms were generally accompanied by some headache and nausea, and followed by a feeling of great weakness and exhaustion, but the patient was always able to go about her household duties in the interval.

A young lady, between twelve and thirteen years of age, who had suffered for about three years from a choreic practice of "making faces," and bobbing her head forwards in a curious manner, was the patient in one of my cases. About three weeks before the date of my first visit (24th June, 1857) she suddenly began to suffer from the paroxysms which have now to be described, and a few months previously she had suffered for some weeks in a similar manner. In one of those paroxysms she would sink or rise into a sitting posture, with her legs folded under her, and then her head would be agitated by a violent, alternating, semi-rotatory movement, until the hair would stream out horizontally on all sides, like the strands of a mop when twirled over the side of a vessel. Then followed a movement in which the whole body was thrown round and round by a succession of rapid vaults. In making these vaults, the hands were placed upon the floor or bed, and the arms used as a kind of leaping-pole; and except at the instant of swinging round, when the feet and legs were thrown horizontally outwards, the half-sitting, half-kneeling posture was never abandoned. The movements of alternating semi-rotation of the head, and of circumvolution of the whole body, occurred separately and without any order, and lasted from a few minutes to half an hour. At their worst the paroxysms were only separated by short intervals; and it is difficult to say whether the movements themselves or the state which followed—a state in which the patient lay panting, dripping with sweat, and exhausted to the last degree—were most distressing to witness. Paroxysms such as these occurred several times a day during the first fortnight of my attendance, and then ceased suddenly. After this the patient rapidly improved in general health, and the choreic twitchings of the muscles of the face and the bobbings of the head became much less frequent. This improvement, however, was only temporary, and at the end of three months the fits returned, though in a modified form, and much less frequently. At this time, indeed, the alternating semi-rotatory movement of the head did not return, and the movement of circumvolution was varied by other movements. Thus, instead of turning, the patient would at times make a succession of leaps in a straight line, so that it was necessary to run in order to prevent her from rushing out at the foot of her bed; and now and then, after falling back exhausted at the end of such a paroxysm, she would roll over and over sideways for three or four times. During these strange attacks there was not the least trace of stupor, and she would often complain of pains in her head, or of being excessively tired even while the muscular disturbance was at its height. In some instances after the relapse, however, her mind was in a rapt or entranced state, and now and then words escaped which showed that she was absorbed by some alarming dream or vision. At those times the eyes had a fixed stare, and the cheeks were somewhat flushed. After the paroxysm she would be for some time in an intensely nervous and excitable state, starting at the slightest noise or the gentlest touch, and now and then bobbing her head with much violence; or, if the mind had been entranced while the movements were going on, this state would continue for some time, and then pass off with a succession of sighs. Ordinarily, however, the mind was perfectly clear, and the first moment of rest was occupied in complaining of the feeling of headache and fatigue from which she suffered. In the intervals, the patient was nervous and excitable, but in every respect an acute, clever, accomplished, amiable girl. At these times her principal complaint was of a dull pain across the top of the head, or of a feeling of tingling in the back and limbs. In this case, the pulse was quick and weak, the hands and feet were habitually cold, chilblains were scarcely absent in summer, anæmic sounds were audible in the heart and great vessels, the appetite was very defective, and the digestion sluggish. There were no worms or any other evidence of derangement in the alimentary canal beyond a slight disposition to tympanitic distension of the abdomen. Nor was there the slightest evidence of uterine derangement; indeed, in this point of view, the patient was a mere child. Recovery was tedious, and more than once interrupted by a relapse, but it was complete in the end; so complete that there was no relapse when menstruation was established about twelve months later.

The next case is that of a young gentleman, Mr. E——, æt. 22, who came up from the country, about six years ago, to consult me for what he considered to be epileptic attacks. These attacks he had, but he also had other attacks, for the sake of which I now refer to the case. In the first place, he had a curious pursing up of the mouth, attended with frequent shruggings of the right shoulder, and frequent tossings out of the right leg; in the next place, he had attacks of shuddering, which were so violent as to shake things out of his hand, or to pitch him bodily out of the chair in which he might be sitting, or even out of the bed in which he might be lying; in the third place, he had what he called a "fit of turning." He had scarcely told me this story, when, after two or three shudders, as if a shock of electricity had been passed through him, he got up from the chair on which he was sitting, and began to turn slowly on his heels upon the hearthrug. He turned round and round in this way perhaps twenty times, and then sat down. Before getting up from the chair he told me not to be surprised at what I saw, and begged me not to attempt to stop him. He said, moreover, that the impulse to turn was not altogether irresistible, but that he could not resist the impulse successfully without being much agitated afterwards. This gentleman had gained honours at college, and there was no reason to conclude that his mental powers were at all impaired. He had suffered for some time from vertigo, and now and then from headache, but never distressingly so. His pulse was 60, and weak, and during one of the paroxysms which I have described it fell full 10 beats, and became much weaker. I noticed, also, that the breathings were slow and embarrassed, and that he drew several long breaths in succession as soon as the paroxysm was over.

It is also customary to regard as varieties of chorea those distressing and not very uncommon cases in which the head is affected by semi-rotatory, oscillatory, bowing, or bobbing movements. These movements are very varied in character and degree: they may be combined in various ways; and not unfrequently one kind changes into another in no very regular or intelligible order. The contractions giving rise to these movements may take place suddenly or gradually; very often they recur with monotonous regularity so long as the patient is awake; in some instances they may now and then be suspended for a time by a strong effort of the will, or by holding the head firmly between the hands: not unfrequently they are accompanied by muscular contractions elsewhere, especially when the patient begins to be worn out by want of sleep and annoyance, and in some degree by bodily suffering also, for, after a time, the muscles affected become very sore, especially about their insertions, and the contraction is attended with a good deal of pain.

Nor does this exhaust the list of affections which have or are supposed to have some relationship to chorea. On the contrary, it remains to mention certain movements which are, often at least, little more than bad habits or awkward tricks, such as semi-uncontrollable winkings, grimacings, and other movements, which are sometimes spoken of as tics-*non*-douloureux. Nay, even stammering, stuttering, giggling, sneezing, and some forms of hysterical coughing, are not excluded, nor yet the convulsive shakings which are often seen in certain paralysed parts, or the jerks and starts which are not unfrequently met with in connexion with epilepsy. In fact, the term chorea is of the widest and loosest significance; for it is scarcely too much to say that it is made to include every form of disorderly involuntary movement, partial or general, which has not altogether the specific characters of tremor proper, or convulsion proper, or spasm proper.

3. PATHOLOGY.—During the last two years Drs. Hughlings Jackson, Broadbent, Tuckwell, Ogle, Barnes, and others, have done much to elucidate the pathology of chorea; the investigations of Dr. Kirkes, made four or five years previously, serving as the starting-point to these new inquiries.

Dr. Kirkes was of opinion that "chorea is the result of irritation produced in the nerve-centres by fine molecular particles of fibrin which are set free from an inflamed endocardium, and washed by the blood into the cavities of these centres;" but he did not venture to fix upon the precise seat of the mischief thus done in these centres. He merely pointed to the vegetations on the valves of the heart which he believed to be constantly present in fatal cases of chorea, and to the signs of heart disease during life in these and other cases, and drew his conclusion.

Adopting this theory of embolism, Dr. Hughlings Jackson goes further than Dr. Kirkes had done, and attempts to prove that the plugging of the vessels, which he regards as the cause of chorea, is in the nerve-tissue forming the convolutions near the corpus striatum—a part supplied by branches of the middle cerebral artery; and that the tissue is thereby not destroyed, but rendered unstable from under-nutrition resulting from a diminished supply of blood. And, without doubt, the clinical evidence adduced in favour of this view is very cogent. Taking chorea of one side of the body, hemichorea, as the simplest form of chorea, and putting it side by side with hemiplegia, the result of embolism, good reason is found for believing that the disorder of movement and the palsy both point to the region of the corpus striatum as the seat of mischief. If this be the seat of mischief in hemiplegia, why not in hemichorea? The muscles most moved in hemichorea are those most palsied in hemiplegia. In hemichorea, as in hemiplegia, the arm, as a rule, is more affected than the leg. In right hemichorea, as in right hemiplegia, the speech is generally very much affected. Again, hemichorea is always more or less mixed up with, and sometimes ends in, hemiplegia; and, on the other hand, hemiplegia from various causes is not unfrequently attended by chorea, or movements of some kind or another. The fact that the face is involved in chorea shows that the seat of the disorder must be above the spinal cord. The facts which have been instanced point to the convolutions near the corpus striatum, rather than in any other part of the brain, as the part affected. In this way Dr. Jackson reasons, and reasons to good purpose; for most assuredly the difficulties which beset any attempt to localize the choreic lesion in the nerve-centres are not a little simplified by thus insisting upon the clinical relations between hemichorea and hemiplegia, as a ground for believing that the region of the corpus striatum is the part affected in both disorders.

Dr. Broadbent also accepts the same doctrine of embolism up to a certain point, and, not knowing that any one had gone before him, travels by the same way to the same conclusion as that which Dr. Hughlings Jackson had arrived at only just before. He is, however, inclined to localize the seat of the cerebral mischief in chorea *in*, rather than near, the sensori-motor ganglia, and he looks upon embolism of the fine vessels of these ganglia only as the chief cause of chorea. As with paralysis, so with chorea, he believes that the symptoms point to the *seat* of the mischief, not to its *nature;* and that, besides embolism, hæmorrhage, softening, irritation, and other causes, may figure among the causes of chorea; the difference between the mischief causing chorea and that causing paralysis being this—that in the one case it is impairment of function only, and in the other case abolition of function—a view which is also insisted upon by Dr. Hughlings Jackson. In addition to embolism as a cause of chorea, Dr. Broadbent instances local innutrition, reflex action from peripheral irritation, and direct action upon the sensori-motor ganglia, from shock, &c. He shifts his ground, in fact, considerably, from embolism as a cause, but at the same time he refers to the discovery by Dr. Bastian of the proximate cause of the delirium of febrile diseases in embolism by altered and cohering white blood-corpuscles, as bringing some of the causes which might be referred vaguely to local innutrition or blood disease within the category of the cases caused by embolism.

Much evidence to the same effect, at least so far as showing that the condition of the heart is favourable to embolism, is also supplied by Drs. Ogle and Tuckwell, though Dr. Ogle himself is not in favour of this theory of embolism.

Dr. Ogle reports sixteen cases of fatal chorea occurring at St.

George's Hospital since 1841, and all taken from the hospital books. In ten of these, fibrinous bands were present on the cardiac valves; and in eight of these ten, their seat was on the auricular surface of the mitral valves. In another case also, not included in these ten, the carotid artery was plugged up. Dr. Tuckwell has witnessed five fatal cases in all of which the valves of the heart were affected in the same way—a way so constant as to lead him to speak of hearts thus altered as *choreic* hearts, the peculiarity being in the presence on the auricular aspect of the mitral valves, along the free margin of each cusp, of a line of numerous, bright, clustering, warty vegetations, some as large as a pin's head, others so minute as to be just visible to the naked eye, and that only in a certain light, but shining like little white beads when slightly magnified, which bodies might be easily detached by lightly brushing the part with a camel-hair brush or with the tip of the finger, and some of which had been detached, and were clinging to the chordæ tendineæ of the valve, ready to pass into the circulation at the next contraction of the heart. In order to find these evidences of valvular disease, as Dr. Tuckwell points out, it is not enough to open the heart in the ordinary manner, and look at the mitral valve from below: for, looked at in this way, the valve may appear quite healthy. It is necessary to slit up the left auricle and look at the valve from above; and because this is not always done, no doubt the disease has been often overlooked when it only wanted looking for to be detected.

The appearances met with, after death, in the nervous system are more difficult to explain in accordance with what has been already said. In a few instances only do they tend to confirm the notion of the choreic lesion being caused by embolism, and localized in the region of the sensori-motor ganglia. In one of two fatal cases, of which Dr. Tuckwell gives the details, mania was the most prominent symptom during life, and the post-mortem examination discovered an extensive red softening of the convolutions—"a consequence of embolism;" and in the other, in which there was no mania or delirium, there was no superficial softening of the grey matter, but a deeper-seated softening of the right, and in a less degree of the left hemisphere, in that part which lies outside and beneath the sensori-motor ganglia, without any recognisable evidence of embolism, the corpus striatum and optic thalamus lying, as it were, embedded in a nest of softened cerebral matter. Dr. Tuckwell also cites a case of embolic hemiplegia, with choreic movements supervening upon the paralysis, in which the same parts were found softened after death.

"In the beginning of May 1860, a girl, aged 19, was admitted into St. Bartholomew's Hospital, under Dr. Burrows, with complete hemiplegia of the left side, and a loud musical systolic murmur at the apex of the heart. The diagnosis made was 'plugging of some cerebral vessel by fibrin detached from a diseased mitral valve.' Within a fortnight from the time of her admission, while the paralysis of the left side was steadily improving, the right side became paralysed, and both the right and left sides became affected with well-marked chorea. On May 27, double pneumonia, involving the right more than the left lung, came on; and she died on May 29. The post-mortem examination was made by Dr. Harris, and I took down the following from his dictation:—The brain was found healthy at all points except at the under part of the middle lobe in either hemisphere, where there was a well-marked patch of softening, about as large as a hen's egg, larger in the left than in the right hemisphere. The brain tissue at the softened part had a reddish-yellow tinge, more marked on the left than on the right side. The middle cerebral artery, on either side, at about its third division, was found obstructed, at an angle of bifurcation, by a firm fibrinous deposit. The heart had its left venticle hypertrophied, and the auricular surface of its mitral valve studded with numerous warty growths. The right lung was partly in the first, partly in the second stage of pneumonia. The upper lobe of the left lung was in the first stage of pneumonia. There were no deposits in the liver, spleen, or kidneys."

Dr. Bastian also refers to a fatal case of bilateral chorea, with delirium, of which he promises to give the details presently, in which embolisms, consisting of masses of irregular shape and size, and evidently made up of an agglomeration of white blood-corpuscles, had led to ruptures and obliterations of small vessels throughout the corpora striata and the course of the middle cerebral arteries generally. So far the appearances in the nervous system after death from chorea agree with the premises, but not so what remains to be stated. Thus, in fourteen cases of deaths from chorea, collected by the late Dr. Hughes, the brain was quite healthy in four, and only congested in three cases, while of the remaining seven cases the particulars are these:—In the first, serous effusion beneath the arachnoid and into the ventricles, slight effusion of blood beneath the right cerebral hemisphere, softened brain; in the second, arachnoid opaque, brain dark and soft; in the third, pia mater watery, cineritious matter, red, soft, and partially adherent; in the fourth, brain soft and vascular, much fluid in ventricles; in the fifth, arachnoid opaque in parts, cerebrum vascular, left thalamus rather soft; in the sixth, dura mater adherent very firmly to calvarium, more opaque than natural, cerebral vessels turgid; in the seventh, blood effused into arachnoid, fornix and edge of third ventricle soft, red, and tumid, brain softened. In the same fourteen cases, the spinal column was not opened in six: of the remaining eight, the cord and its membranes were quite healthy in three, and only a little congested in one; and of the four others, the particulars are these:—In the first, soft adhesions of the arachnoid, grey matter dark; in the second, vessels rather large and numerous, serous surfaces opaque, old adhesions of the membranes, especially posteriorly; in the third, medulla slightly softened, rachidian fluid opaque, yellow, and densely coagulable by heat; in the fourth, softening of the cord opposite the fourth and fifth dorsal vertebræ. Nor is the information supplied by Dr. Ogle in the paper already referred to less vague, for the sum of it is only this—that the brain or cord, one or both, were more or less congested in six cases, that the central parts of the brain were much softened in one, and that the cord was softened in one and otherwise affected in another. Very possibly a different result might have been arrived at if these cases had been examined with special reference to the condition of the sensori-motor ganglia, especially if more men like Dr. Bastian were concerned in the investigation. As it is, all that can be said is that the facts of morbid anatomy do not supply *much* support to the notion that the choreic lesion is caused by embolism and localized in the sensori-motor ganglia. There is nothing in the facts to contradict the notion that the choreic lesion may *begin* in the sensori-motor ganglia: there is something to show that all parts of the nervous centres may become affected in the end—the cord as well as the brain. No doubt, as Dr. Reynolds remarks, the symptoms of chorea point from, rather than to, the cord. No doubt the spasm should be tonic rather than clonic, as it is in chorea, if the cord were specially at fault in chorea. No doubt the cessation of the spasm of chorea in sleep points to the brain, which does sleep, away from the cord, which does not sleep. Nor are these the only reasons which point away from the cord; but the fact remains that after death from chorea the cord is often found to be affected, and also that a particular part of the cord, the posterior columns, is specially affected in a disease which agrees with chorea in this, that it is marked by inco-ordination of movement, namely, locomotor ataxy.

Neither do the teachings of experimental physiology help much towards exactly localizing the particular mischief which operates in the exceptional cases of chorea. These teachings show that movements of a rotatory character may originate in various parts of the nervous system—in the thalami optici, corpora quadrigemina, crura cerebri, pons Varolii, crura cerebelli, in certain parts of the medulla oblongata, and also in the upper portion of the spinal cord; that choreic agitation may be caused by slicing away the cerebellum, and by puncturing one of the corpora quadrigemina; that the removal of the encephalon in front of the thalami optici *may* result in an impulse to go forwards; and that a deep wound in the cerebellum *may* be attended by an impulse to go backward. "The parts injured," says Dr. Brown-Séquard, "seem to be quite different from those employed in the transmission of sensitive impressions or of the ideas of the will to the muscles, at least in the medulla oblongata and pons Varolii. They constitute a very large proportion of these two organs, perhaps three-fourths of the first one: they are placed chiefly in the lateral and posterior columns of these organs: they seem to contain most of the vaso-motor nerves, by which, directly or through a reflex action, they may act on other parts of the nervous system: and they can give rise to spasm on the *same* side of the body—a fact which shows that many of their fibres do not decussate." Moreover, another lesson to be learnt from experimental physiology is, that rotatory movements may have their starting-point in a *nerve* at a distance from the nervous centres. Thus, Dr. Brown-Séquard has made a rabbit turn or roll towards the injured side by puncturing the expansion of the auditory nerve within the ear; and M. Flourens has produced similar movements in a pigeon, by simply tying a bandage over one of its eyes. It would seem, indeed, as if the parts of the nervous centres which are concerned in the production of choreic movements may be affected from a distance by *reflex* action. Nor is this to be wondered at, seeing that there are facts without number which show that distant parts of the nervous system are continually being affected by reflex action, and that the varied consequences of a particular injury are only to be accounted for by supposing many of them to be reflex phenomena. Indeed, there is no lack of instances to show that any part of the nervous system may act on any other part, and the exact localization of many disorders of the system is a difficult if not hopeless task, for the simple reason that any given lesion in any part may be attended by a wide range of symptoms depending upon sympathetic disorder set up in other parts.

At first sight it may be supposed that the pathological facts which have been given, favour the idea that inflammation of the brain or spinal cord, one or both, has to do with the production of chorea; but a moment's reflection is sufficient to dispose of this supposition. It is plain, in fact, that this inflammation cannot be regarded as essential to the chorea, for in some of the cases there are no traces of inflammation. This inference is inevitable. Moreover, the clinical history of these very inflammations, apart from chorea, leads to the same conclusion, for the symptoms of these inflammations are not those of chorea. There are also on record many cases in which inflammation in other parts, as in the lungs, has been developed in the course of chorea, and in which the choreic symptoms have been suspended during the inflammation. The case, indeed, is one which seems to justify the inference that the chorea is connected, not with inflammation, but with a state which may issue in inflammation. The case is one in which all seems to be explained if it be supposed that the chorea is connected with *irritation*, not with inflammation—with the state, that is to say, which precedes inflammation always, and which may or may not issue in inflammation. In this way, then, the cases which have been given, in which the traces of inflammation are absent after death, must be looked upon as cases in which the chorea proved fatal before irritation issued in inflammation, and the cases in which the signs of inflammation were present, as cases in which before death the irritation had issued in inflammation. Nor is there anything contradictory to this conclusion in the clinical history of the cases of which the post-mortem appearances were those of inflammation, for there is nothing in this history to show that this inflammation may not have occurred *very shortly* before death, and that the true choreic symptoms may not have disappeared as the true symptoms of inflammation made their appearance.

And, certainly, there is little reason for connecting chorea with fever. On the contrary, there appear to be good grounds for believing that the maxim of Hippocrates holds good here as in other cases—*febris accedens solvit spasmos.* At any rate, there are many cases on record of measles, scarlet fever, rheumatic fever, or some other fever, being developed during the course of chorea, and in which the choreic symptoms have been suspended during the fever. I have met with seven such cases. Indeed, so far as I have had the opportunity of judging, the constant rule appears to be, that the chorea is aggravated in the initial stage of the fever—that is, in the cold stage, or stage of irritation—and suspended more or less completely when the stage of reaction, or hot stage, is established; and that, in relation to rheumatic fever, the place of chorea is either before the fever (often a long time before) or after the fever (often a long time after).

The history of chorea in relation to inflammation and fever, indeed, so far as I can see, would seem to be like that of disorders which are more or less akin to chorea—namely, tremor, convulsion, and spasm in their various forms. For what is this history? In an attack of common trembling, the circulation is greatly depressed, and the pulse does not recover itself until this paroxysm is over; and in paralysis agitans the paleness and chilliness of the surface of the body, and the decided relief afforded by wine, tell a similar story. In delirium tremens the cold perspirations, the quick and fluttering pulse, the moist and creamy tongue, are all significant facts. The initial rigor of fever, moreover, is coincident with defective surface-warmth, miserable pulse, sunken countenance, blueness of nails, cutis anserina, and other signs of vascular collapse, and subsultus goes along with the most utter prostration of the powers of the circulation. And in mercurial tremor, an inference as to the real state of the circulation may be drawn from the fact that the subjects of this disorder are not unfrequently in the habit of resorting to gin and other stimulants for the purpose of making themselves steady. There even appears to be something uncongenial between tremor and an excited state of the circulation. The state of the circulation in the delirium of which trembling is the distinctive feature—delirium tremens—is quite different from the state of the circulation in the delirium in which there is no trembling. In the latter case—in the delirium of acute meningitis, for example—the skin, especially the skin of the head, is hot and dry, not cold and damp; the pulse is hard and strong, not weak and fluttering; the tongue is parched and brown, not moist and creamy—the condition is one, in fact, of high fever, and not one which, as in delirium tremens, is more akin to collapse than to high fever. And it is not less certainly a fact, that delirium tremens loses its characteristic trembling if acute head-symptoms and high fever make their appearance in the course of the disorder. Moreover, it must be borne in mind, as pointing to the same conclusion, that the initial rigors of fever disappear *pari passu* with the establishment of the vascular reaction of the hot stage, and that they return in the form of subsultus when the state of reaction has died out, and the patient is left utterly prostrate and helpless. Again, there is reason to believe that spasm is associated frequently with a depressed state of circulation. During the attack of catalepsy, the appearance of the patient is very like that of a corpse, and it may even be necessary to apply the ear to the chest to know of a certainty that the heart continues to beat. In tetanus, as all are agreed, there is no fever; and in the tetanus arising from strychnia, as Dr. Harley has shown, one effect of the poison is to prevent the blood from becoming properly oxygenated. In cholera the cramps are coincident with a state of almost pulseless collapse. In hydrophobia, the condition of the circulation is as far removed from feverish excitement as in tetanus. And, certainly, a similar inference may be drawn with respect to the state of the circulation in cramp in the leg and elsewhere, for these seizures are met with, not in strong persons, but in those who are weakly, and especially in those who are elderly as well as weakly. Nay, there is reason to believe that spasm in its various forms is antagonized rather than favoured by an excited state of the circulation. In tetanus it appears to be the rule for the spasm to gain ground almost in exact proportion to the degree in which the pulse loses its true power. In hydrophobia it would seem as if the same law held good, for on analysing the histories of a considerable number of cases, I find that there was less agitation, less convulsion, less spasm, where the circulation was less depressed than it is in the ordinary run of cases. Nor is a different conclusion to be drawn from the history of spasm as it is set forth in whooping-cough. For what is the fact? The fact is simply this—that the whoop, which is the audible sign of the spasm, does not make its appearance until the febrile or catarrhal stage has passed off; that it disappears if pneumonia, bronchitis, or any other inflammation be developed in the course of the malady; and that it returns when the inflammation has departed. And most assuredly there is no clinical evidence to show that convulsion is associated with an over-active condition of the circulation. In the fevers of infancy and early childhood, especially in the exanthematous forms of these disorders, convulsion not unfrequently occupies the place which belongs to rigor in the fevers of youth and riper years. It occurs in the cold stage of the fever, when the powers of the circulation are greatly depressed in every way; and it is confined to this stage, except in those cases in which there are certain brain and kidney complications, when it may also take the place of subsultus, or rather of death itself, for when it occurs at this time the patient has all but ceased to strive in the "struggle called living." Nay, I am even disposed to think that there is something altogether uncongenial between convulsion and the hot stage of the sympathetic fever connected with inflammation, for it is a fact not unfrequently verified that fits of common epilepsy are often suspended for the time by causes which give rise to a state of sympathetic fever in the system. For example, I can call to mind four or five cases of epilepsy, in which high sympathetic fever was set up by a burn or other injury inflicted during a fit, and in which fits, which were of daily occurrence before the accident, and which recurred with the same degree of frequency afterwards, were altogether suspended so long as the fever continued. Nor is a contrary conclusion to be deduced from the history of the convulsion connected with teething, with worms, or with any other condition in which what is called "morbid irritability" is the prominent characteristic; for it is found, not only that fever is almost entirely foreign to the state of "morbid irritability," but also that convulsion, when it does occur, is associated with seasons of decided vascular depression. In a word, the result of bedside study has been to convince me that the true place of convulsion, in connexion with any form of febrile disorder, is in the cold stage before the hot stage, or in the cold stage after the hot stage, and not in the hot stage itself; that, in fact, there is something uncongenial between convulsion and an excited state of the circulation. And so also with ordinary epilepsy, the general history of the disorder appears to be that the convulsion is antagonized by an excited state of the circulation rather than favoured by it.

As it seems to me, then, there is nothing unintelligible in the fact that chorea, instead of being connected with a state of inflammation and fever, is connected with a state which must be looked upon as the very opposite of inflammation and fever. As it seems to me, indeed, there is nothing in this part of the history of chorea but what was to be expected from the history of tremor, convulsion, and spasm in their various forms. Nay more, the antagonism between chorea and inflammation or fever is, as it seems to me, nothing but what is necessitated by the physiological as well as by the pathological history of muscular contraction. But these are topics upon which I may not dilate further in this place, and I therefore bring my remarks under the present head to a close, by simply saying, that those who care to know more of what I think on this subject will find the latest statement of my views in a book about to be published under the title of "The Dynamics of Nerve and Muscle."

4. CAUSES.—"The patients who suffer from chorea," says Dr. Hillier, "are very impressible and emotional, and very liable to derangements of the nervous system." Often too, as in the case which has been given as an example of the disease, where there were fits at the time of teething, they have suffered from some other decided disorder of the nervous system, and quite as frequently; as also is illustrated in the case in question, where the mother had epilepsy at the time of puberty, an inherited disposition to disorder of the nervous system may be suspected. Thus, out of 48 cases in which I have inquired into the family and personal history of the patients, I find 27 cases in which father or mother, or brother or sister, had been, or was, subject to epilepsy, paralysis, apoplexy, hysteria, chorea, or insanity; and 11 in which the patient had had infantile convulsions, chorea, or epilepsy. As in hysteria, it might be expected that sympathy and imitation would figure among the causes of chorea; but this anticipation does not appear to be borne out by the facts. "The disease,"

says Dr. Hillier, "is never induced by the assemblage of several choreic patients in a ward of children, nor does it appear that the symptoms are in any way aggravated by mutual association." Fright, on the other hand, is without question a frequent cause; it is distinctly stated to be the exciting cause in 31 out of 56 cases collected by Dufossé and Bird, in 34 out of 100 cases reported by Hughes, in 9 out of 31, and in 9 out of 38, related by Dr. Peacock and Dr. Hillier respectively. Still it is certain that in many of these very cases the *début* of the choreic symptoms is so long deferred as to make it difficult to believe that fright has had very much to do as an exciting cause, and not unfrequently also a doubt as to the operation of any sudden exciting cause is suggested by the very slow development of the choreic symptoms. Indeed, when the matter is strictly inquired into, but few cases are to be met with in which the patient was at once suddenly sent into a state of chorea from any cause, emotional or other. Some special cause of *irritation* may also be suspected in some cases, as worms in the intestines, a fœtus in the uterus, and especially unnatural irritation of the sexual organs; but here again the evidence is less conclusive than it might be supposed to be. One or two cases are on record in which choreic symptoms have ceased almost abruptly on the expulsion of a tape-worm by a vermifuge. Chorea in pregnancy has also been found to cease on delivery. Still it may be questioned whether chorea in pregnancy is always true chorea, and whether an altered state of the blood rather than irritation may not be the true cause in those cases where there is no good reason to be in doubt as to the nature of the disease; for in pregnancy there is a hyperplastic state of the blood which may favour embolism, from the direct deposit of white corpuscles in the minute vessels, as is pointed out by Dr. Bastian, if not by the floating into these vessels of minute vegetations detached from the cardiac valves. In 5 out of 16 cases of fatal chorea recorded by Dr. Ogle there were "proofs of congestion and other graver lesions of the genital system;" and most certainly I have in not a few cases found reason to know or suspect the existence of practices which might lay the foundation of such congestion, and give rise to any amount of irritation, and this too in cases where the age of the patient might be supposed to be a sufficient contradiction to the notion. Again, the causes of chorea and rheumatism would seem to be closely allied, if not identical. "Chorea," says Dr. Tuckwell, "is a disease which is common among the poor and ill-nourished, rare among the rich and well-favoured; and exactly the same holds for rheumatism." Chorea also resembles rheumatism in being more common in damp and cold than in warm and dry climates. At the same time, season does not seem to influence the development of chorea very much. Thus, in 27 cases given by Dr. Hillier, 13 occurred in the six winter months and 14 in the six summer months—viz., 2 in January, 5 in February, none in March, 3 in April, 2 in May, 4 in June, 2 in July, 0 in August, 2 in September, 1 in October, 3 in November, and 2 in December.

5. Diagnosis.—In a well-marked form, chorea cannot well be confounded with other maladies. It does not even suggest the idea of hysteria, and therefore there need be no confusion on this score. Nor need chorea be confounded with inflammatory diseases of the brain and spinal cord. In the more aggravated cases there is, I believe, a tendency to run on into one or other of these diseases, and the moment of transition may not always be easily definable; but, as a rule, the accession of the new disease will be indicated, not by the aggravation, but by the cessation of the choreic symptoms proper, and the substitution for them of delirium, pain in the head, convulsion, paralysis, numbness, pain in the back increased by movement, and others, in groupings which will leave no doubt as to what their true meaning must be. I have seen two cases of chorea in children which ended in cerebral meningitis, and in which the choreic movements ceased when convulsion and delirium made their appearance; and I have seen one case of severe chorea in a youth which ended in inflammatory disorganization of a considerable portion of the spinal cord, and in which the choreic symptoms did not continue after the development of the numbness, paralysis, and other symptoms of myelitis. Indeed, it may be stated broadly that the symptoms of chorea are *not* the symptoms of acute inflammatory affections of the substance of the brain and spinal cord, or of their meninges. Delirium is reckoned among the symptoms of acute chorea in some cases. But I am very much disposed to believe that the case has changed from chorea to some other disease of the brain, not always inflammatory, of course, when delirium makes its appearance; and that this case will be spoken of, not as chorea, but as a consequence of chorea, when more is known of the diagnosis of diseases of the nervous system, and when greater exactness of nomenclature is attained to. Nor need chorea be confounded with any chronic affection of the membranes or substance of the brain or cord, the points of difference to be noted being always more numerous than the points of resemblance. In locomotor ataxy the disorderly movements are mostly in the legs, and in these parts only when attempts are made to stand or walk; whereas in chorea the movements, which are chiefly in the upper part of the body, though aggravated by any attempt to use the will, continue at all times with little or no intermission so long as the patient is awake. In chorea also there are none of the severe neuralgic pains which are so characteristic of locomotor ataxy. Again, chorea is emphatically what locomotor ataxy is not—a disease of childhood. The history of chorea is also sufficiently distinct from that of the jerks and shocks attending epilepsy and paralysis agitans. In the former case I have sometimes seen movements so repeated, and so like those of chorea, that a mistake might have been possible if the fact of the fits had been overlooked; but usually the movements attending epilepsy are jerks and shocks, separated afterwards by wide intervals and extending over a long period, and not at all choreic in themselves. The history

of paralysis agitans, let alone the age of the patient, is sufficiently distinctive. And so is the history of those cases of paralysis complicated with choreic movements, in which paralysis is the primary disorder, and which may be spoken of as a sort of local paralysis agitans; for this history points to previous brain-disease in a way not to be mistaken, and to a time of life which is in itself inconsistent with the idea of chorea. In fact, it is scarcely possible to confound ordinary chorea with any disorder of the brain or cord, acute or chronic, if only moderate care be used in the diagnosis. And this is all that need be said under this head; for with respect to the exceptional forms of chorea, general or partial, it is more than probable that they ought to be taken out of the category of chorea and placed with hysteria, or referred to some special disease or disorder of the brain or cord.

6. PROGNOSIS.—The natural tendency of chorea is, without doubt, towards recovery. Sooner or later, as a rule, the patient gets well; and too often, as it would seem, the treatment deserves very little credit for this result. The mean time occupied in recovery, according to Sée, is 69 days, or a trifle under 10 weeks; and Dr. Hillier, basing his calculations on 30 cases treated by himself, arrives at the same conclusion, the longest time occupied by these cases in recovery being 28 weeks, the shortest two weeks, and the mean 10 weeks. The disposition to relapse is considerable, and usually primary attacks are more protracted than relapses. Now and then, in the proportion of six per cent. according to Sée, chorea takes an acute form, and is rapidly fatal. Local chorea, as exhibited in the muscles of the neck at least, is notoriously obstinate; and instead of wearing itself out, it is more likely to go on year after year until the patient is worn out by it. How far the occurrence of chorea implies a tendency to other disorders of the nervous system, especially to epilepsy, is a question which has not yet been fully entertained, and I cannot supply an answer from actual statistics. But this I may say—that I have *frequently* met with epileptic patients who were choreic at one period of their life, and that the impression left on my mind from what I have seen is, that the chances of chorea being followed, sooner or later, by some other disorder of the nervous system are too much made light of.

7. TREATMENT.—Nothing can be more perplexing than the statements made by various authorities respecting the efficacy of remedial agents in the treatment of chorea. Few voices, it is true, are now raised in favour of the old-fashioned antiphlogistic ways of treatment, in which bloodletting and purgatives and low diet figured so conspicuously; but beyond this all that is uttered seems to be dictated by the spirit of contradiction or scepticism. Indeed, so little unanimity of opinion is there respecting the treatment which ought to be pursued in chorea, that the only course is for each one to

glance at the principal remedial agents recommended, to weigh the statements made respecting them as well as he can, and to take upon himself the responsibility of deciding upon his own course of action.

Sir Thomas Watson considers that the most suitable medicine in cases of chorea is, as a rule, some preparation of *iron;* and this verdict is accepted by the great majority of English practitioners in medicine. Dr. Elliotson says that he cured forty cases in succession by the use of full doses of sesquioxide of iron, the time spent in the cure varying from six to eight weeks. I have not used iron much in the treatment of chorea, and I have not seen it used to any great extent by others. Not unfrequently, however, I have known a person using this agent go on for a while with it, and then discontinue its use, apparently as if he were not satisfied that all the good was being done which ought to be done. Of the several preparations of iron which have been recommended, I am disposed to believe most in the syrup of the iodide, the use of which was first suggested by the late Dr. Barlow, of Guy's Hospital. I have certainly seen several cases in which the use of this preparation seemed to be followed by unequivocal evidence of improvement; but, on reflection, I find it difficult to refer this change for the better to the iron altogether, or even to the iron chiefly. On the contrary, I am disposed to think that the iodine is entitled to a fair share of the credit, to say the least; and that the iodine in the doses usually given is stimulating or restorative in its action rather than alterative, in the sense in which it is usually supposed to be alterative. I fancy, also, that there is a growing doubt as to the efficacy of iron in cases of chorea, and that many would now be disposed to agree with the late Dr. Hughes, who says only that iron has been administered in numbers of the cases of chorea recorded in his admirable report, and that it has "sometimes succeeded where zinc has failed."

Zinc is given very largely in the treatment of chorea. In the cases of chorea occurring in Guy's Hospital, the late Dr. Hughes says that "zinc in the form of sulphate has been the most frequently employed as a remedy, and has generally been most successful;" and forty-five cures out of sixty-three cases, or five in seven, are credited by him to this medicine. Dr. Barlow says, "In ordinary cases, the exhibition of purgatives to keep the bowels freely open, and the sulphate of zinc in doses gradually increased from a grain to even fifteen or twenty grains, or even more, will effect a cure. When, however, the sulphate has been used in these large doses, its sudden discontinuance seems to be felt by the system, and a return of the symptoms ensues. The best rule, therefore, for its exhibition is as follows:—the bowels being kept open, the sulphate of zinc should be given in doses commencing with a grain three times a day, and in the case of a child about twelve years old the quantity should be increased by the addition of a grain daily, until the medicine causes sickness, or there is an obvious diminution of the choreal movements. In the former case the dose should be diminished by at least one-half, and so continued for several days, with a view to establishing a tolerance; but if, on the other hand, there be a marked improvement, it should be no further increased, but continued without alteration until either the improvement ceases—in which case it should be again gradually increased—or the disease has altogether subsided. Whenever the latter is the case, we ought to diminish the dose day by day, rather than discontinue it suddenly, as by following the latter course we have less reason to dread a relapse." In continuation of these remarks, Dr. Barlow adds, "In some cases, however, especially those in which there is considerable anæmia, the iron seems to have more control over the disease than has the zinc, though these cases are rather exceptionable ones." The late Dr. Bright tells us that he found the sulphate of zinc answer where the carbonate of iron had failed, and that where iron succeeds, there the zinc had done no good; and Sir Thomas Watson, who repeats this statement of Dr. Bright, leaves us to infer that these words express his own experience in the matter.

Arsenic is another favourite medicine in the treatment of chorea, especially in Great Britain and Ireland. Thomas Marten was the first to recommend it, now sixty years ago; and since his time it has been very extensively used. Dr. Romberg, speaking of the various remedies recommended, and alone deserving confidence as capable of arresting the disease in a short space of time, says, "The foremost among those an experience of several years has taught me to be arsenic." Dr. Begbie also writes, "In an experience of nearly thirty years I have never known arsenic fail." Nor would it be at all difficult to cite other authorities to the same effect. Dr. Begbie gives five drops of Fowler's solution twice a day, an hour after a meal, and adds a drop to the dose every day until the specific effects of the mineral upon the system are observable, and then he suspends the treatment for a while. He goes on with the medicine, that is to say, until he is warned to stop by itching and swelling of the eyelids, by redness of the conjunctivæ, by a white, silvery appearance of the tongue seldom accompanied by tenderness, and by nausea and uneasiness at the pit of the stomach. I have often used arsenic in the treatment of chorea, and I have great faith in its efficacy as a medicine in the malady. At the same time I have often abandoned its use in consequence of the gastric disturbance which, do what one will to prevent it, was set up by it. It seemed, indeed, as if in these cases the stomach would not tolerate the medicine in doses large enough to produce a sufficiently rapid action in the cure of the disease. It did not follow, however, that this intolerance of the stomach was a sufficient reason for abandoning the arsenic in these cases, for the stomach is not the only channel by which this medicine could have been introduced into the system. Failing the stomach, indeed, the hypodermic or endermic method might have been tried, and that too, I have now reason to believe, with many chances of advantage to the patient. The case which suggested to me the hypodermic use of arsenic was that of a patient in the Westminster Hospital (Hallett Ward), Margaret S—— by name. This patient had suffered for nine years from a distressing choreal affection of certain muscles of the neck, by which the head was kept continually turning and bobbing. At different times various modes of treatment had been tried, including the hypodermic injection of morphia and atropine, without the least benefit. When first admitted under my care, and for the three weeks following, I gave her bromide of potassium and morphia, my chief object being to procure sleep and alleviate pain in the neck; for the muscles in the neck, which were the seat of the morbid movements, were very tender in many places, and the movements themselves attended with much pain; but harm, rather than good, seemed to be done by these means. The idea of injecting arsenic hypodermically occurred to me on the 12th of January 1866, and was carried out on the same day. Fowler's solution was chosen, and the part selected was the most tender point over the contracting muscle. Three minims were injected on the 12th, ♏v. on the 15th and on the 27th, ♏vj. on the 19th, ♏viij. on the 22d, ♏vij. on the 25th, and again on the 29th, ♏viij. on February 1st, ♏ix. on the 3d, ♏x. on the 6th, ♏xj. on the 8th, ♏xij. on March 1st, and again on the 10th, ♏xiij. on the 12th, ♏xiv. on the 14th. On the 21st the patient left the hospital almost well. Before the fourth injection was practised a marked change for the better had taken place; before the eighth the choreal movements were almost at an end, and the change for the better had gone on steadily progressing from the beginning. Between the eighth and the ninth injection there was an interval of three weeks[1]—the injections being suspended on account of the local irritation and inflammation which they had set up. When the patient left the hospital there was some stiffness in the muscles which had been the seat of the disturbance, by which the head was slightly twisted, and the voluntary movements of these muscles were not free; but every day there was a change for the better in these respects. In the hospital, the only treatment associated with the injections was a gymnastic one, the patient being made to move her head from side to side, and backwards and forwards, in time with a slowly moving pendulum, together with an occasional dose of morphia at bed-time, this drug being given less on account of the malady in the neck, though pain in this region was still complained of at night, than on account of a distressing habit of sleeplessness. Two months have now elapsed since the patient left the hospital. She occasionally presents herself for inspection, and her state continues very much the same as it was, just one step from being quite well, and not bad enough to make her wish to have the injections repeated. She goes on exercising the muscles of the neck with the pendulum, and having them shampooed, and for medicine she has now and then had some cod-liver oil. In this case the object in introducing the arsenic hypodermically was, not to escape gastric irritation, but to produce some local change in the nerves of the parts which were the seat of the disorder, as well as to bring about some more general change in the system. I have employed, with results more or less satisfactory, the hypodermic injection of arsenic in several analogous cases, and also in certain cases of neuralgia, epilepsy, and other affections of the nervous system; and thus the case which I have given is not the only case which furnishes to my mind reason for believing that this mode of treatment may be of use in the treatment of certain cases of chorea. I have also used arsenic endermically as well as hypodermically in a few cases of chorea. In order to this I have dropped from fifteen to twenty drops of Fowler's solution upon lint moistened with water, and applied this, under oil-silk, night and morning, to a raw blistered surface. This application gives rise to considerable local irritation; indeed, it generally, before the week is over, has the effect of covering the blistered surface with a thin, dry eschar, and of causing a zone of angry pimples to crop up in the skin immediately surrounding the part which has been blistered. Owing to this irritation, indeed, it is generally necessary to make pauses in the treatment after going on for six or seven days at a time. As yet, however, I have little practical experience of the effects of this mode of treatment in actual cases of chorea. I have tried it in two cases of average severity, in one of which the patient was well in twenty-eight days, in the other in thirty-two days; and this is all that I can say respecting it except this, that as with the hypodermic method before mentioned, so also with this, I have given it a trial in certain cases of neuralgia and epilepsy, and that the results arrived at in these cases lead me to hope that this mode of giving arsenic may prove to be a not unimportant addition to the *armamenta therapeutica.* With respect to the comparative merits of the hypodermic and endermic methods of introducing arsenic into the system I cannot yet speak. I incline to give the preference to the former method, both as least distressing to the patient and as most efficacious; but I have, as yet, no sufficient practical experience to justify the expression of a definite opinion.

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[1] Up to the eighth injection undiluted Fowler's solution was used; when the injections were resumed, and after this time, this solution was diluted with an equal quantity of water. In other cases, also, where the same mode of treatment has been carried out, I have employed a mixture of equal parts of this solution and water, for I found that the solution diluted to this extent produced very much less local irritation than the undiluted Fowler's solution.

During the last twenty-five years *strychnia* has been employed somewhat extensively in the treatment of chorea, especially in France. Dr. Trousseau was the first, or among the first, to do this, and after an experience of a quarter of a century he is still disposed to give the preference to this practice. The preparation employed by this physician is a *syrup of the sulphate of strychnia,* made by dissolving 3 grains of the sulphate in ℥x of simple syrup; and the manner of giving it, which is peculiar, is as follows:—In children from five to ten years of age, the treatment is commenced by giving a *teaspoonful* of this syrup (containing $\frac{1}{28}$th of a grain) twice or thrice a day—one dose in the morning, another in the evening, and the third, if there be a third, at noon. On the next day these doses are repeated. On the following days, each day an additional teaspoonful of the syrup is given until six teaspoonfuls are given, care being taken to distribute these four, five, or six doses at equal intervals through the day. Having arrived at this point, if the physiological effects of the dosing are not yet produced, *dessertspoonful* doses are substituted for teaspoonful, and the same rule is observed with these larger doses as with the smaller. Beginning with two or three dessertspoonfuls in the course of the day, and giving three on the next day also, the doses are increased by a dessertspoonful each day, until six dessertspoonfuls are taken in the course of the day, care as before being taken to distribute these doses, few or many, with intervals between them as wide as possible. If the desired effect be not yet produced, a still bolder practice is pursued, and a tablespoonful of the syrup is substituted for *one* of the dessertspoonfuls; and you are to go on, still augmenting, but in a way which is not very clearly laid down. "En augmentant progressivement," M. Trousseau says, "avec la même prudence, avec la précaution essentielle de distribuer le médicament à des intervalles sensiblement égaux dans le courant de la journée, vous arrivez à donner aux enfans de cinq à six ans 50, 60, 80 et jusqu'à 120 grammes [5 grammes go to the teaspoonful] 25 milligrammes de sirop; 3, 4, jusqu'à 6 centigrammes de sulfate de strychnia." In persons older than ten years, Dr. Trousseau begins with large doses, with dessertspoonfuls in place of teaspoonfuls, and goes on until he reaches 200 grammes of the syrup—a quantity containing no less than 10 centigrammes, or 1½ grain, of the active principle. The object is to produce the full physiological effects of strychnia, and to maintain them for a while, and the duration of the treatment is said to be thirty-three days for girls and seventy-four days for boys. When the medicine begins to tell upon the system, the symptoms are, twenty minutes after taking it, or thereabouts, slight stiffness in the jaw or neck, some headache, confusion of sight, and giddiness, and some disagreeable "démangeaisons" in the parts of the skin covered with hair. Afterwards as the system becomes more deeply impressed, the stiffness extends from the jaw and neck to the limbs and elsewhere, especially to the limbs most affected with chorea, which limbs are also in all probability more or less paralysed, the itching of the skin is no longer confined to the hairy parts, and painful jerks or shocks, or still more obvious tetanic symptoms, make their appearance. The tolerance of strychnine varies not only in different individuals, but in the same individual at different times, so that the dose which was not more than enough one day may be poisonous the next. In fact, the treatment is one which requires to be most carefully watched, and which cannot well be watched with comfort, especially by the friends of the patient, however enlightened or forewarned they may be.

Another heroic treatment for chorea, which has found some favour in France, is that by *tartar emetic*. Laennec has left on record three cases of chorea treated by large doses of this medicine, and others have tried the same method with results, as they seem to think, more or less satisfactory, especially MM. Boulay, Gillette, and Henri Roger M. Gillette's method, which is that adopted by M. Roger, is to give the antimony for three days, to withhold it for three or five days and again to give and withhold it for the same period, as often as may be necessary, if the symptoms have not yielded to the medication of the first three days. On the first day of the first triple series of days, the dose given in the twenty-four hours is from 20 to 25 centigrammes (1 centigramme is = ·15, or nearly $\frac{1}{7}$th of a grain). This dose is doubled on the next day, and tripled on the third day; then the patient is allowed to rest from three to five days. On beginning again, if this be necessary, the dose given on the first of the three days, which is to be doubled on the next day, and tripled on the day following is 5 centigrammes larger than that used on the day in which the treatment was commenced. If this be not enough, after waiting again for from three to five days, the dose for the first of the three days is 5 centigrammes larger than that used on the first of the last series of three days, for the second day the dose is doubled, and for the third day the dose is tripled; so that, if the dose given on the first of these three days was 30 centigrammes, the dose on the last of these three will be 90 centigrammes, *or nearly* 14 *grains!* In the majority of cases we are told the first doses are followed by nausea and vomiting of a glairy matter, but these symptoms soon pass off, and complete tolerance is established, especially if care be taken to withhold as much as possible all dietetic drinks. We are told also that diarrhœa is uncommon, that constipation is not uncommon, that the pulse becomes slower, that the skin moistens, and that the general health improves. Indeed, Dr. Bourguignon, speaking of certain children, patients of M. Gillette, under this mode of treatment in the Hôpital des Enfans Malades, at Paris, says: "Les enfans ne sont nullement abattus, ils conservent leur gaieté." Dr. Bourguignon, who is strongly in favour of this treatment, tells us also that in ten cases—whether in M. Gillette's practice, or in his own, he does not say—the patients got well in sixteen days, as an average, the shortest time being four days, the longest twenty-four.

Iodide of potassium is another remedy which has been tried somewhat extensively in the treatment of chorea, and to a less extent so has *bromide of potassium.* This iodide was supposed to be indicated by the probable existence of a rheumatic or lymphatic predisposition in the patient, or by the actual presence of some meningeal irritation or inflammation, and these indications have been carried out fully and frequently; but the practical results of this treatment, so far as I know, are unsatisfactory. Nor is a different opinion to be expressed with respect to the bromide. I have tried this medicine in several cases, and tried it fully, and from what I knew of its strange efficacy in epilepsy I was strongly prejudiced in its favour; but the result, as I have said, is that the bromide appears to be no more justly deserving of confidence than the iodide.

As might be expected, *opium* is a medicine which has not been overlooked in the cases in question, especially in the severer cases. As in tetanus, there appears to be a remarkable tolerance of this medicine in chorea, and in several cases enormous doses have been given; indeed, in any case it appears to be necessary to give large doses, in order to procure what may be supposed to be the object in view, that is, sleep. I have seen opium employed in five very severe cases of chorea, largely, and from what I saw in these cases I am not wishful to see the experiment repeated. I am speaking now of the free use of opium by itself, and not of opium in moderate dose along with other agents, with the free use of alcoholic stimulants especially. This, I believe, is quite a different matter. Nor does there appear to be sufficient reason for supposing that other narcotics, not excepting *cannabis indica*, are more to be trusted than opium in casea of chorea.

The *inhalation of chloroform or ether* has been had recourse to in many severe cases of chorea. I have seen three such cases in which chloroform was used in this way, and my impression was that harm, not good, was the result. I believe, also, that harm rather than good is likely to be done in these cases, unless alcohol is given in sufficient quantities before the inhalation. If this be done, the patient may remain asleep for some time, and awake the better; if this be not done, there is great danger, so far as my experience of the use of chloroform inhalations is concerned, of the patient waking almost immediately, and of being more unnerved and more agitated than he was before he was put to sleep. At the present time I am in attendance upon a case of chorea, attended with much sleeplessness, until the practice was adopted of giving at bed-time a few whiffs of chloroform *after* a glass of hot negus. The chloroform had been tried for four nights without the negus, and harm rather than good had been the result. It has now been tried with the negus for a week, and, as it would seem, with unmistakeable advantage. Nor is this an isolated case.

Antispasmodics, such as camphor, ether, valerian, assafœtida, and musk, have been tried extensively, and the general verdict appears to be that they are not useless. I am disposed to place considerable confidence in *camphor*, and also in *ether;* in camphor especially. I often give very generally this last-named medicine dissolved in cod-liver oil, and my impression, from what I have seen, is that this addition to the oil is a decided advantage to the patient.

Turpentine has been given for various reasons in chorea—as an anthelmintic and purgative chiefly. At one time I gave it rather as a general stimulant, and, as it seemed, with benefit to the patient. I then tried *mineral naphtha* with the same view, and came to the conclusion that this medicine was more pleasant than turpentine, less trying to the system, and not less efficacious. During the last six or eight years, however, I have rarely given either one or the other of these medicines, and one chief reason for this seems to be that I have gradually come to prefer the treatment of which I have to speak in a few moments.

Ammonia is also a remedy which has some good claim to be mentioned in the present place. I have tried the sesquicarbonate in several cases, singly and in combination, and the trial has been to my mind eminently satisfactory. I am, for example, at present seeing a little choreic boy who had been for three weeks treated, without any benefit, with sulphate of zinc, and who has wonderfully improved during the last three days, by leaving off the zinc, and by substituting sesquicarbonate of ammonia in five-grain doses every three hours. In other respects there was no change in the treatment, and the patient is too young to allow it to be supposed that he was affected beneficially by the *change* of the practitioner.

For various reasons, theoretical and practical, the free use of *alcoholic drinks* has long seemed to me to be the foundation of a rational plan of treatment in chorea, and the larger experience of the last few years has only served to confirm me in this opinion. I have seen enough to know that, as a rule, the change for the better is unmistakeable when, after the carrying out of a contrary mode of practice, alcoholic drinks are given with a liberal hand. I have notes of three cases of great severity, where rapid amendment was brought about by giving, at frequent intervals, an egg beaten up with a large glass of sherry or with an equivalent dose of brandy, and I verily believe that this plan would rarely fail if carried out *in time*—if carried out, that is to say, before the nervous system had become thoroughly exhausted and broken down, as it does do in the end. Indeed, in a bad case, where a dangerous degree of sleeplessness had to be dealt with, there is nothing in which I should have more confidence than in the free use of alcoholic drinks. I should look upon these means properly, that is freely, used as the natural means of procuring sleep and all the beneficial consequences of sleep. I should be afraid of attempting to attain the same end by the use of *medicines* more or less analogous to alcohol in their action, because these medicines would all of them be more likely to disturb the action of the stomach, and so interfere with the restoration of the system by food. And for the same reason I should even be almost afraid of giving small doses of opium with the view of conciliating sleep, though I have no doubt that the proper dose of this drug at the proper time, in conjunction with the proper dose of alcohol, might be very satisfactory practice. In a word, I cannot but think that it is a perfectly rational way of dealing with severe cases of chorea to push alcoholic drinks until they produce drowsiness—until, that is to say, they exercise a decidedly sedative action upon the system. At any rate I have carried out this idea in more than one case of the kind with what seem to me to be very satisfactory results.

In bad cases of chorea, as a matter of course, the *recumbent position* is a necessary part of the treatment; and in cases of ordinary severity my own impression is that the patient would improve more rapidly if he were kept longer in bed. Indeed, it surely stands to reason that *rest*, properly used, is a right means of remedying a state of muscular disorder, in which muscular fatigue is an unmistakeable element. Nay, it is not too much to imagine, that the persistency of many cases of chorea may be not a little owing to the patient being allowed to be up and fidgeting about when he ought to be in bed.

Exercise, on the other hand, properly used, cannot well be dispensed with as a means of treatment in chorea. In ordinary cases, indeed, it is difficult to overrate the importance of suitable gymnastics as a means of cure. This is no new idea. Darwin insisted upon it long ago ; and from what has been done in this direction since his time, especially by Ling and his successors in the practice of the so-called "movement cure," it is perhaps not too much to say that chorea may be one of the consequences of neglecting gymnastics as a means of education in children. Certain it is, that ordinary cases of chorea get better rapidly—the average duration of the period of treatment being sixty days—under a properly arranged course of gymnastics, with little or nothing else. The practice of M. Sée, at the Hôpital des Enfans Malades in Paris, may be cited in support of this statement; and it would not now be difficult to find corroborative passages in the practice of others. For myself, I should think that I was omitting an important duty if I did not prescribe the use of some suitable exercise for a patient suffering from chorea—the use of a skipping-rope or trapeze, if nothing else. Dancing has long been a favourite idea with me as a means of exercise in cases like these ; and so have calisthenics regulated by music. More than one choreic patient I have known to be cured by learning to dance, and I think that music might be employed with advantage now, as it was in the case of the tarantula dance of old, in quieting severe cases of chorea, anomalous or not. Indeed, there is more than one case on record in which music has been so employed. It may be supposed also that music will help the choreic patient in his gymnastic efforts in the same sense as that in which it nerves the acrobat to the performance of his wonderful feats. In a word, it is not necessary to think long before it must become self-evident that orderly movements, be they those of dancing, calisthenics, or more special gymnastics, and be they regulated by music or not, are natural remedies for disorderly movements such as are met with in ordinary cases of chorea, and that a very important means of cure is neglected if they are not provided. Indeed it is to be hoped that the time is not far distant when a suitable gymnasium will be considered as much a part of the proper fittings of a hospital as the dispensary, and when medical men more generally will be alive to the importance of suitable gymnastics, not only as an educational, but also as a curative measure. Surely there is a lesson to be learnt from the results of the carrying out of the "movement cure,"—a lesson which the practitioners of orthodox medicine are not justified in continuing to decline to learn because it happens to have heterodox belongings !

Baths, of one kind or another, have been extensively employed in the treatment of chorea. In this country the *cold shower bath* has been the favourite mode of bathing, and there are some good grounds for this preference. Part of the good results is ascribed to the shock; part—a greater part, perhaps—to the reaction. Still there are, unquestionably, many cases in which the shock is not tolerated, and where reaction is not easily established—cases in which the patient is rendered worse rather than better, so far as the chorea is concerned, with the additional disadvantage of a bad cold, or actual rheumatism, or some other evil. And these latter cases are by no means uncommon. Nay, it may even be suspected that all cases would come into this category if care were not exercised more or less. The same remarks apply also to cold plunge baths, and to other forms of cold baths. With respect to *hot baths* and to *warm baths*, the case is very different. A hot bath at bed-time has often seemed to me to have a marked calmative influence. I am also disposed to think that a good part of the benefit ascribed by M. Baudelocque to *sulphur baths* (each bath contains about four ounces of sulphuret of potassium) is to be ascribed to the high temperature of the water, or, at any rate, to this in conjunction with the counter-irritation set up by the action of the sulphuret upon the skin. The fact appears to be that baths of one kind or other are not sufficiently recognised as a means of cure, not only in chorea, but in many other cases of disease, by the orthodox practitioners of medicine. With baths, indeed, it is very much as it is with "movements" as a means of cure, and hydropathy, like kinesitherapy, has a lesson to teach, which medical men ought to set themselves to learn if they would be fully provided with the means by which to contend successfully against disease.

Electricity is another agent which requires a passing mention in this place, though all that can be said respecting it is, that as yet there appears to be little or no reason for placing any confidence in it as a means of treatment. Whether this will be always the case—whether there are not modes of using electricity which will have the effect of quieting choreic and analogous movements (so long, at any rate, as they are used)—remains to be seen. I suspect that there are such modes, and that they will be beneficial, and that too not a little, in the case in question, but I have not yet the *facts* to justify the expression of a belief on the subject.

For the last seven years I have employed cod-liver oil in many cases of chorea, and, so far as I can judge, I have good reason to be satisfied with the results. In adopting this practice my main object was to restore nerve-tone by improving the nutrition of nerve-tissue. I remembered that fatty matter was an essential ingredient in nerve-tissue; and, remembering this, I came easily to the conclusion that one natural way of attaining to the end in view was to take care that the food contained a sufficient amount of fatty and oily matter. Without a due supply of these matters, I reasoned, the nerve-tissue must be of necessity starved—that, in fact, to withhold these matters, or to supply them in insufficient quantity, would be as great a mistake in cases where the object was to improve the nutrition of the nerves, as it would be to withhold lean meat in cases where the object was to get more muscle. I argued in this manner, and be the theory right or wrong, I think, as I have said, I have no reason to be dissatisfied with the results of putting it in practice.

For the last seven years also I have used phosphorus in the majority of cases of chorea in which I have used cod-liver oil, and for the same reason. I asked myself whether the fact that phosphorus is present in large quantity in the great nerve-centres, and that the amount of this ingredient seems to have some direct relation to the activity of the nervous functions, being as much as 2 per cent. in adult life, and below 1 per cent. in infants and idiots, might not show that phosphorus is specially indicated as food for a weak nervous system—as much indicated, perhaps, as iron in cases where there is a deficiency of red-corpuscles in the blood; and this question once put seemed to require an answer in the affirmative. "In small doses," says Dr. Pereira, "phosphorus excites the nervous, vascular, and excretory organs. It creates an agreeable feeling of warmth in the epigastrium, increases the fulness and frequency of the pulse, augments the heat of the skin, heightens the mental activity and the muscular powers, and operates as a powerful sudorific and diuretic." In large doses, without doubt, phosphorus is a caustic poison; in proper doses, it produces the very changes which are to be desired in cases of chorea and analogous forms of convulsive disorder. In proper doses and properly watched, it is quite innocent in its action, and may be very beneficial. Of this I am confident. The forms in which I first gave phosphorus in chorea were the phosphorated oil of the Prussian Pharmacopœia and the ethereal tincture of the French Codex (forms containing 4 grains of phosphorus in the fluid ounce), but lately I have preferred the hypophosphites, especially the hypophosphite of soda, for the simple reason that these salts, which were originally recommended by Dr. Churchill of Paris as specifics in phthisis, are infinitely less nauseous than the oil or tincture, and not less efficacious. I have given for some time from 5 to 8 grains, three times a day, of the hypophosphite of soda to children, in cases of chorea, without any harm certainly, and, as I think, with unmistakeable benefit, and I have not yet found any reason to change this practice for another.

In an ordinary case of chorea, the plan of treatment which I have now adopted as a rule for some time is to give cod-liver oil in conjunction with hypophosphite of soda, making the draught containing the latter salt the vehicle for the administration of the cod-liver oil. With these medicines, according to circumstances, I have associated camphor or ammonia, one or both, adding the sesquicarbonate of ammonia to the draught containing the hypophosphite, and *dissolving the camphor in the cod-liver oil.* I have found that this latter solution is an excellent way of giving the camphor, and also that the camphor masks the taste of the oil not a little, and makes the stomach more tolerant of it. I have not kept notes of all the cases which I have treated in this manner, but I think I am quite within bounds when I say that the number now amounts to upwards of sixty, and that the average duration of the treatment was under one month.

I may also add that I have in three or four cases given arsenic along with hypophosphite of soda and cod-liver oil, and that the result, to say the least, was not such as to discourage a continuance of the practice.

If there be any special sources of irritation, as worms or carious teeth, these of course must be met and dealt with. If the agitation be so great that there is danger of the skin being excoriated, or of the patient falling out of bed, properly padded sides must be fixed to the bedstead, or it may be expedient to encase the body and limbs of the patient in cotton-wool. If the affection be confined to certain muscles of the neck or elsewhere, it may be expedient to use hypodermic injections of arsenic, as in the cases related in the text, to divide a nerve, as has just been done by Mr. Campbell De Morgan, or to use the actual cautery. If there be a morbid mental condition, as there too often is, moral means of treatment must not be neglected. In fact, each case of chorea must be looked upon from a special as well as from a general point of view, and the success of the treatment will, in many instances, if not in all, depend upon the skill with which special means can be combined with those general means of which I have spoken, and upon which I have prosed at greater length than I ought to have done.

PARALYSIS AGITANS.

By William Rutherford Sanders, M.D., F.R.C.P.

Synonyms.—Paralysis tremens, tremula, jactitans, palpitans; Tremor artuum, T. coactus; Scelotyrbe festinans, seu Festinia (Sauvages); Synclonus tremor, S. ballismus (Mason Good); Schüttelähmung, Schüttelkrampf, Zittern; Tremblement sénile; Trémulence paralytique progressive; Chorea senilis, Ch. festinans; Pseudo-chorea, Pseudo-paralysis agitans; Dystaxia agitans; the Trembles, &c.; the Shaking Palsy (Parkinson).

Definition.—Idiopathic Paralysis Agitans consists of involuntary tremulous or shaking motions of the limbs, head, or trunk of the body, which take place even when the parts are supported and unemployed. The voluntary movements are preserved, but their vigour is lessened in the affected parts. In certain, usually advanced, cases, there is a disturbance of equilibrium; most commonly a disposition to stoop, or bend the body forwards, and to pass, in locomotion, from a walking to a running pace. The senses and intellect are unimpaired.

The definition includes these principal characters: 1st, The shaking or tremors, of a spasmodic kind, which occur even when the parts are not in use (Tremor coactus, palpitatio, παλμός, agitatio, jactitatio, quassus); 2d, The diminished muscular power (Paralysis, paresis, pseudo-paralysis); 3d, The disturbed equilibrium, shown usually in the tendency to stoop and to move forwards with accelerated speed (Scelotyrbe festinans, festinatio, procursus). Of these characters the clonic tremors or shaking are the most constant and distinctive. The paralysis, on the other hand, is of a peculiar kind. As here employed, the term does not mean cessation or interruption of voluntary motion, which, on the contrary, persists; but it is intended to designate both the imperfection of the movements, which results from the interference of the tremors, and also the impaired strength and the slowness of muscular action, which are usually observed in the tremulous parts. Some writers, objecting to call this condition paralysis (a name apt to mislead), have spoken of it as "apparent but not real paralysis," or "paresis," or "pseudo-paralysis," or "dystaxia." Lastly, the disturbance of equilibrium does not always occur: it is often late in appearing, and it serves chiefly to mark a special form or an advanced stage of the affection. Nevertheless, in fully developed examples of Paralysis

Agitans, all these symptoms, the tremors, paralysis, forward stoop, and accelerating walk, are associated together; as Parkinson expressed it, there is a combination of Tremor coactus and Scelotyrbe festinans.[1]

Historical Notice.—From the definition, as explained, it will be apparent that the older descriptions of Paralysis Agitans are to be sought less in the history of palsies than in that of spasmodic nervous diseases. In fact, Paralysis Agitans has been overlooked principally from being confounded, 1st, with tremors in general; 2d, with chorea; 3d, with cases of motor palsy (hemi- or paraplegia) complicated with spasmodic and tremulous movements.

1st. As a symptom, tremors early attracted attention. They were briefly noticed by Hippocrates and Celsus, while by Galen and succeeding writers their kinds, their causes, and their value as prognostic signs were fully discussed. At length, nosologists established the genus Tremor, dividing it into species, in some of which tremor was still regarded as a symptom merely, while in others it was recognised as a substantive or idiopathic disease. Accordingly, well-marked cases of simple Paralysis Agitans are found in many of the older authors,[2] by whom they are sometimes alluded to, sometimes described with graphic details, in illustration of the pathology of tremor or as examples of a distinct species of disease. Less notice was taken of the disposition to stoop and hasten onwards. The earliest mention of this curious symptom is probably made by Gaubius; it was first particularly described by Sauvages under the name of Scelotyrbe festinans. But Sauvages did not connect it with tremors; he, indeed, had seen only two cases of it. It is certain that the combination of persistent tremors and hurrying gait had not been recognised, and no adequate description of Paralysis Agitans existed previous to Parkinson's "Essay on the Shaking Palsy in 1817." His account still remains the standard authority. Succeeding authors have, in general, simply quoted it, or have (especially French writers) overlooked the disease altogether. Accordingly, although Parkinson drew attention to the imperfection of our knowledge, the original contributions made since his time have been few and fragmentary. A list of references will be found at the end of this article.

2d. In regard to the confusion of Paralysis Agitans with chorea, it must be remembered, that convulsive diseases have been imperfectly discriminated, owing partly to the difficulty of their pathology, partly to the superstitions with which they have been associated. The disease now commonly called chorea was not so named originally, nor was it confounded with true St. Vitus's dance; it was regarded merely as a kind of convulsion (motus convulsivus) or species of epilepsy (epilepsia gesticulatoria), till near the end of the 17th century.[3] About that period, Sydenham, in the brief description which fixed the characters of the disease, unfortunately named it "Chorea Sancti

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[1] Parkinson seems to consider the festination as essential to Paralysis Agitans, bu cannot be so regarded.

[2] Sylvius, Bonet, Juncker, Van Swieten, Sauvages, &c.

[3] Roth.

Viti," a misnomer which it has since commonly retained, with the effect of confounding it with the dancing mania, from which it is quite distinct.[1] While older authors therefore may have described cases of Paralysis Agitans among the motus convulsivi extraordinarii (just as some authors have given definitions applicable to it under the name hieranosos),[2] it is only in recent times that Paralysis Agitans has been confounded with what is at present known as chorea. This confusion is due partly to a certain similarity in the diseases, partly to the unsettled state of medical nomenclature. Ordinary cases of the shaking malady are widely distinct from common chorea; but certain extreme forms occasion a violent irregularity of movement, resembling in a great degree the gesticulations of that disease; so much so, that it has been proposed to regard Paralysis Agitans as a more intense form of chorea,[3] and cases of Paralysis Agitans have been recorded under the title St. Vitus's dance.[4] Some cases even exhibit a combination of Paralysis Agitans and chorea.[5] Moreover, while the common gesticulatory chorea is well known, there are other rarer forms of irregular and uncontrollable spasmodic movements, as yet imperfectly studied and classified, to which the term chorea is usually applied: such are the rotatory or spinning-top chorea, the saltatio or leaping ague, malleatio, &c. In some respects, therefore, it is not altogether inappropriate to designate Paralysis Agitans as a species of abnormal chorea; hence some recent authors employ the terms chorea senilis and chorea festinans for Parkinson's disease. The objection to such names is, that they tend to confound Paralysis Agitans with the ordinary St. Vitus's dance, from which it is entirely different.

3d. Lastly, the term Paralysis Agitans or shaking palsy has been applied, both before Parkinson's essay and since, to cases of ordinary motor paralysis (hemi- and paraplegia) complicated with tremors—a complication not uncommon both in diseases of the brain, and especially in certain cases of chronic myelitis and of locomotor ataxia. Etymologically the name of shaking palsy belongs perhaps to these, rather than to Parkinson's disease, but time has consecrated his use of the term. Parkinson's malady is *Idiopathic* Paralysis Agitans, in which the tremors or shaking are the chief and earliest symptom, and the paralysis entirely subordinate and peculiar, true hemi- or paraplegia being rare complications: while, in the cerebral and spinal affections just referred to, the loss of motion (akinesia) or sensation (anæsthesia) is the main feature of disease, and the tremors and spasmodic agitations are only concomitants (*i.e.* the Paralysis Agitans is *Symptomatic*).

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[1] Authors distinguish the common chorea of Sydenham as chorea minor, the dancing mania as chorea major, choreomania, or tarantismus. By chorea or St. Vitus's dance, however, Sydenham's disease is now always meant, the tarantism from its rarity being left out of account.

[2] Linnæus, Gen. Morb., Upsal, 1763, p. 17, No. 144: "Hieranosos, Byting, Corporis agitatio, continua, indolens, convulsiva cum sensibilitate." Also Vogel and Macbride.

[3] Eisenmann, remark on Dr. Haas's case, in Canst. Jahrb. 1852, iii. 92.

[4] Trousseau, case in 1843. See references.

[5] Maclachlan.

Hence the latter class of cases should be styled, not Paralysis Agitans, but hemi- or paraplegia, or spinal or cerebral disease *complicated with Paralysis Agitans: i.e.* with spasmodic tremors. This distinction, which is essential for the accurate definition of Parkinson's disease, has often been overlooked, and requires, therefore, to be specially insisted upon.

In the following description of Paralysis Agitans, besides some allusion to tremors generally, it is proposed for the sake of distinctness to recognise certain subdivisions of the disease, which the experience accumulated since Parkinson's essay seems to require.

Divisions.—Idiopathic Paralysis Agitans is divided into I. General (including the bilateral and unilateral), and II. Local.

I. General Idiopathic Paralysis Agitans presents certain forms or varieties important to distinguish, as regards prognosis and cure :—

A. Senile forms. *Paralysis Agitans senilis,* occurring in advanced life, above fifty or sixty; usually incurable and with fatal tendency from senile decay: divided into 1st, *Simplex,* and 2d, *Festinans* or *Procursoria.* Varieties, *Unilateralis* or *Hemiplegica* and *Retrograda.*

B. Non-senile forms, occurring under fifty, without fatal tendency, sometimes curable. 1st, *Paralysis Agitans non-senilis, simplex* (*i. e.* sine festinatione), including also *hysterical* and *reflex* Paralysis Agitans, &c. 2d, *Paralysis Agitans toxica,* including, chiefly, tremblement métallique, mercurial palsy, &c.

The curable forms have been supposed to be *Functional;* the incurable, *Organic.*

It will be necessary to describe the senile forms in detail, as they are much the most frequent; a shorter notice will suffice for the others.

Description.—I. A. Idiopathic Paralysis Agitans senilis. 1st, simplex, when attended by the signs of senile decay only; 2d, festinans, or procursoria, &c., when the disturbance of equilibrium is also present. These two forms will be described together.

Symptoms and Course.—Onset usually gradual; course slow, progressive, liable to be arrested at different stages; duration protracted; associated with senile decay. Several stages may be recognised.

1st Stage. Commencement.—The first symptoms are usually so insidious that the patient cannot tell precisely when they began. A sense of weakness and a disposition to trembling is felt in some part, most frequently the hand or arm, sometimes the leg or head. The tremors, at first slight and occasional, gradually increase; and at an uncertain period, seldom less than a year, the corresponding parts of the opposite side, more rarely the other limb of the same side, become affected. The tremors and muscular debility seldom extend beyond the arms during the first two years, which period may be said to comprise the first stage. Except for the inconvenience arising from the unsteadiness of the hand in writing or other manipulation, the patient would not consider himself the subject of disease. At this period, probably, remedies might be applied with success. In a few cases, instead of the ordinary gradual approach, the tremors have come on rapidly after a fright or exposure to cold.

2d Stage. Generalization of the Tremors.—Some time after the hands and arms have been affected, one of the legs, usually that on the side first attacked, begins to tremble and is more easily fatigued; and in a few months the other leg becomes similarly tremulous and weak. Walking becomes a task requiring considerable attention. The legs feel heavy as lead, and are not raised to the height or with the promptitude which the will directs, so that care is necessary to prevent frequent falls. At a later period, usually some years after, the tremors extend to the head, and finally to the whole body. The tremors of the limbs are usually in the direction of flexion and extension, sometimes of rotation, sometimes of ab- and adduction (so that patients have had their knees padded to prevent them knocking together). In the head and neck the movements are more commonly lateral (shaking negatively), then vertical (nodding). The lower jaw is affected with vertical, rarely lateral motions; and the tongue is tremulous, impeding speech: in many cases, however, these parts are not affected till near the end of the disease. The larynx is little, if at all, affected. Deglutition does not suffer till near the close. The muscles of the eyeballs and eyelids, and the facial muscles of expression, are nearly always exempt from tremors.[1] The thorax and trunk are later and less affected than the limbs, or head, or neck. Appearing chiefly during a general paroxysm of tremors, the spasmodic action of the respiratory muscles occasions a peculiar panting of the breathing and a jerking interruption of speech. As the tremors last, and become general over the body, they increase in intensity; from mere vibrations they become violent convulsive agitations. The limbs are jerked to and fro as if by the action of springs or by rapid shocks of electricity.

From the beginning, and throughout the whole course of the disease, mental emotion or agitation excites an attack of tremors, or greatly aggravates them; rest and quietude diminish or stop them. In general, a slow, firm, voluntary act, or the grasp of a heavy body,[2] stops the tremors for a time, and any change of posture has the same effect, affording the patient considerable relief. Parkinson mentions an artist, who, while his arm and hand were palpitating strongly, would seize his pencil, with the effect of instantly suspending the tremors and allowing him to use it for a short period.[3] On the contrary, when the limbs are quiescent, a voluntary movement usually starts the tremors, which continue for some time afterwards. The attacks of tremors are at first of short duration, and separated by intervals of complete immunity; they become more severe and the intermissions shorter as the disease proceeds. In certain examples the paroxysms have lasted so long as ten to forty minutes, and were followed by fatigue like that produced by violent muscular exercise.[1] The tremors cease entirely during sleep.

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[1] In Oppolzer's remarkable case the tremors are reported to have extended to the muscles of the face; also in a few other cases.

[2] A patient we saw lately holds a smoothing-iron to keep his hand still; another steadies it by seizing a chair.

[3] Lebert refers to a similar instance.

Parkinson does not seem to have noticed that frequently, in addition to the tremors, there occur tonic spasms (rigidity or contraction) of the muscles in the parts affected. The fingers or toes or the whole limb become rigidly flexed or extended. These cramps last for some minutes, and return at intervals; they are sometimes painful and followed by a sense of fatigue. They occur chiefly during the day, but sometimes in the night also. In a case recently observed by the writer startings of the limbs took place during sleep, in the form of powerful flexion of the knees, by which the legs were suddenly drawn up. On the relaxation of the spasms, the limbs were slowly let down to their previous position without awakening the patient.

Local deformities also sometimes result. From the hands being kept constantly supported to stay the tremors, the fingers become dislocated backwards on the metacarpals so as to form an angle with the back of the hand.[2] Sometimes the distortion is lateral; in a case lately seen the fingers were bent obliquely to the radial side, owing probably to the clonic and tonic contractions being more powerful in that direction. These deformities must not be confounded with the effects of rheumatism.

3d *Stage. Disturbance of Equilibrium.*—The occurrence of this symptom is variable: sometimes it appears early, while the legs are becoming tremulous; sometimes it is deferred for ten or twelve years or more after the tremors; in many cases it is entirely absent (Paralysis Agitans simplex). It is therefore less a stage than a peculiar feature characteristic of one form, or of a special extension of the disease; its presence should accordingly always be indicated by some additional term, such as *festinans* or *procursoria.*

Owing to deficient power in the extensor muscles of the back, the patient becomes less able to preserve the erect posture; he bends forward while sitting, still more while standing. In walking, the centre of gravity being displaced forwards, while the legs can only be moved slowly, stiffly, and with some degree of spasmodic jerking and agitation, he is in constant danger of falling; he stumbles over small obstacles in his path, and by taking short, hurried steps he is impelled from a walk to a run, till he has difficulty in stopping himself. Persons in an early stage of this condition can sometimes march slowly with long measured strides, quite well; but so soon as they resume their shuffling gait, they must quicken their pace to avoid falling. There is no vertigo, as in cases of precipitancy from tumours or injuries of the cerebral peduncles and adjoining parts. The forward running is the usual form of this curious symptom, which has not yet been much studied, but exceptional varieties occur. Thus Romberg met with an opposite disturbance of balance. "Two patients, aged sixty, felt a constant desire to walk or fall backwards, and therefore carried the head strongly bent forwards: one of them, in order to stand, separated his legs widely, at the same time crossing his arms on the back, with the view of offering some resistance to the overpowering tendency to move backwards" (Paralysis Agitans retrograda). Graves mentions a patient who had to be balanced to and fro before starting, and who, if arrested in his forward movement, immediately began to hurry backwards and could not stop himself. No case is recorded of a disposition to fall or move sideways.[1] There is a less degree of this symptom in which the patient stoops and shuffles in his walk, but has not the true festination.

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[1] Trousseau, Clin. Méd.

[2] Ibid.

4th Stage. Disease fully established.—When the tremors have become general, violent, and of frequent recurrence, the patient experiences great inconvenience, which increases with the progress of the disease. The limbs cannot execute the directions of the will in the common offices of life. The patient is unable to write or perform any manipulation: he cannot hold a book to read, and has the utmost difficulty or is quite unable to clothe or feed himself. Raising a glass of water to the lips is impossible; the fluid is spilled and the glass knocked to and fro against the mouth. Patients deprived of assistance, in order to allay their thirst, have lapped up fluids with the tongue, like the lower animals. It is painful to witness the struggles which the patient makes to control the agitation of his body and effect some desired movement; the more he tries the more extravagant the jactitations become. To increase his distress, paroxysms of tremors now often arise during rest; indeed, at times, the tremors become almost constant, with frequent aggravations. Commencing for instance in one arm, the wearisome agitation is borne until beyond sufferance, when by suddenly changing the posture it is for a time stopped in that limb, but commences generally in less than a minute in one of the legs or in the arm of the other side, often spreading over the whole body. Harassed by the tormenting round, the patient has recourse to walking, to which he is partial, both on account of the relief afforded by change of posture and because his attention is diverted from his unpleasant feelings by the care and exertion required for its performance. But if the procursive tendency has appeared, this relief is denied. The propensity to lean forward becomes invincible. Forced to step on the toes and fore-part of the feet, while the upper part of the body is thrown forwards, he is irresistibly impelled to take quick and short steps, and to adopt unwillingly a running pace, in order to avoid falling upon the face.[2] On some days, however, the tremors are less severe; and the patient is always relieved by intermissions during the day and complete cessation of the tremors during the night. The disease, even at this stage, sometimes undergoes remissions for some weeks or months, during which the tremors greatly abate: unfortunately a relapse occurs and the disease resumes its progressive course.

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[1] Sauvages relates of a painter, aged 50, that he was not only impelled forward in walking, but could not turn right or left till he stopped himself against an obstacle, supported by which he turned his body gradually round and then hurried straight on anew. This is given as Scelotyrbe festinans, without any mention of tremors; but similar conditions have been observed in Paralysis Agitans.

[2] In the words of Trousseau, "Il s'en va trotillant, sautillant,—*il est obligé de courir, pour ainsi dire, après lui-même.*"

5th. Advanced and Final Stages.—Hitherto the jactitations have been suspended at intervals during the day, and have ceased entirely at night. But in this stage tremors of the limbs occur even during sleep, and increase till they awaken the patient, often with much mental agitation and alarm. In addition, signs of failing strength and physical decay, which had previously appeared, rapidly increase. Unable to convey food to the mouth, the patient must be entirely fed by others. The bowels, previously torpid, require powerful stimulating medicine or mechanical aid for their relief. The trunk becomes permanently bowed, and the whole muscular power fails. The patient walks with great difficulty; a stick no longer suffices; he requires an attendant, who, walking backwards before him, prevents his falling forwards by the pressure of his hands against the fore-part of his shoulders. His words are scarcely intelligible, and the memory and intellect are weakened. The actions of the tongue and pharynx are so hindered by enfeebled action and perpetual agitation, that the food can hardly be masticated or swallowed; the saliva mixed with particles of food continually drains from the mouth.

Finally, amid increasing general debility and diminished voluntary power, the tremors become more vehement, and seldom cease for a moment. When exhausted nature seizes a small portion of sleep, the motions become so violent as to shake the bed-hangings, and even the floor and sashes of the room. The chin is bent down upon the sternum; the power of articulation is lost; the slops with which he is fed trickle continually from the mouth. The urine and fæces are passed involuntarily; bed-sores form; and at the last constant sleepiness, and other marks of extreme exhaustion, usher in the fatal termination.

The senile forms of the Paralysis Agitans, as just described after Parkinson, represent, it should be observed, the most aggravated examples of the disease. And the subjects of it being advanced in years, the effects of senile decay are necessarily mixed up with the other symptoms. Indeed, this kind of Paralysis Agitans seems to induce and to terminate in general failure of the system. But the course of the senile disease is not always so deplorable. Many cases of the simple or non-procursive form remain stationary for an indefinite period, and never reach the ultimate stages.[1] A few exceptional cases have even been cured. The procursive Paralysis Agitans also occasionally exhibits an arrestment, or, at least, extreme protrac-

[1] Dr. Maclachlan, out of a large number of instances among the inmates of Chelsea Hospital, found that the affection often had little or no influence in shortening life. None of his cases had occurred below 55, the majority between 65 and 70, yet in many the disease lasted upwards of 30 years. An in-pensioner, in his 107th year, had been affected since he was 60.—Page 213.

tion of its course. The fatal forms seldom occupy less than ten years. At the same time, Parkinson's account, drawn directly from nature, represents, without exaggeration, the slow, continual progress and the fatal results of the senile Paralysis Agitans in its full development.

One very important fact, observed in nearly all cases of Paralysis Agitans, is that the cutaneous sensibility is not affected, either in regard to pain, touch, or temperature. The sensory powers, indeed, persist remarkably even amid the general failure of nervous energy in the later stages of the disease.

The *Unilateral*[1] or "*Hemiplegic*" variety of Paralysis Agitans, first noticed by Marshall Hall, presents no essential difference from the bilateral (paraplegic) disease, just described. The limbs on one side are agitated with violent chronic tremors, while those on the opposite side are entirely unaffected, or exhibit only a slight and occasional tendency to tremble. The affection is not less severe than the bilateral, into which it probably passes. There is no complete case of this form, from beginning to end, on record. In one instance, lately under observation,[2] there was no disturbance of equilibrium, no festination, and it does not appear that this symptom has been met with in the unilateral disease. The prognosis is, probably, the same as in the other senile forms.

To sum up: Paralysis Agitans senilis occurs in advanced life, past fifty, usually past sixty years of age; it is combined with and appears to hasten senile decay. Two forms of it are distinguished, the *Simple* and the *Procursive* (festinans): it is usually very protracted, lasting ten years or more, and is, with rare exceptions, incurable.

We pass now to those forms of the disease which occur earlier in life.

I. B. Non-senile forms of Paralysis Agitans, occurring under fifty, without fatal tendency, and sometimes admitting of cure. They are much rarer than the senile forms of the disease.

1st. *Paralysis Agitans simplex, non-senilis* (*sine festinatione*).—This form resembles the senile disease in regard to the tremors, differing chiefly by the absence of the signs of senile decay. The jactitations affect the same parts, the limbs, head, and trunk, exempting the muscles of the eyeball, and usually also the facial muscles of expression.[3] They come on in paroxysms excited by attempts at voluntary movements, or by mental emotion; they subside or disappear during rest, and they cease entirely during sleep. In severe cases they are extremely violent. The limbs and the whole body quiver and shake convulsively in the most extravagant manner. The patient cannot stand without support; in walking he jerks and staggers as if moved by broken springs, and is like to be pitched to the ground at every step. He cannot dress or feed himself; if his limbs are approximated, they knock against each other; and if his hand is brought near the chest or the face, it strikes upon them in a series of quickly repeated blows. When the shaking arises unexpectedly, the patient may hurt himself, knocking his head against a wall, &c. It is rare, however, for the tremors to exhibit such extreme vehemence; more commonly they exist only to the extent of rendering the execution of regular movements impossible, interfering completely with the patient's usefulness. It is further observed that, although sometimes as severe, or more so, than in the senile forms, the tremors are less continuous and never occur during sleep, although they may come on as soon as the patient awakes or turns in bed. The special distinctions of the non-senile Paralysis Agitans are therefore: 1st. There is no disturbed equilibrium; no disposition to fall or hasten forwards or backwards.[1] 2d. The diminution of voluntary muscular power is slight: sometimes none is observable. Tested by the grasp of the hand, by the dynamometer, or by the ability to lift weights or bear burdens, the muscular force is often found equal to the normal standard; sometimes the shaking arm appears stronger than the sound one. Yet the patient himself usually complains of diminished strength, and he has less ability to sustain prolonged exertion. If the disease progresses, the muscular debility increases. This is an important sign, for increasing muscular weakness is of unfavourable prognosis. 3d. There is no fatal tendency. The affection is extremely obstinate, often incurable, but the general bodily health is not impaired, and the duration is indefinite if no complication supervene. A patient, lately seen, aged sixty-six, was first affected at twelve years of age; the tremors have entirely unfitted him for labour during nearly his whole life; yet even now his appetite and bodily health are excellent. Similar cases are not very rare; but, being regarded as examples of incurable infirmity,[2] they are not brought under the notice of the physician, and probably suffer irremediably from neglect of care at the earliest stages. 4th. Occurring in middle life (twenty-five to fifty), however formidable in appearance, it is susceptible of amelioration, and sometimes of cure. A case was cured by Elliotson by the use of carbonate of iron (1827), and several examples of recovery have been recorded under different methods of treatment.[3] Others, however, have resisted treatment altogether. To account for the fact that some cases are curable while others are not, it has been supposed that the former are functional and the latter organic.

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[1] "Uni-" and "bilateral" are preferable to "hemi-" and "paraplegic," being less apt to lead to confusion with ordinary motor paralysis.

[2] Patient of Dr. Warburton Begbie, to whom the writer is indebted for an opportunity of examining it.

[3] Marshall Hall mentions a male, aged 28, with Paralysis Agitans of right arm and leg, who presented a "peculiar rocking motion of the eyes, and a degree of stammering and defective articulation." Certainly, however, tremors of the oculo-motor muscles are very rare in Paralysis Agitans; singularly so, since nystagmus by itself is not uncommon.

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[1] At least no case of non-senile Paralysis Agitans, accompanied by festination, is known to the writer.

[2] Often objects of charity, or paupers in or out of workhouses. The disease, however, affects the rich as well as the poor.

[3] Trousseau, case of St. Vitus's dance (properly Paralysis Agitans) in 1843; Russell Reynolds; Handfield Jones; Sanders, case of dystaxia or Pseudo-paralysis Agitans. This patient, after a year, was able to return to light work, the tremors having nearly ceased. Dr. Alexander Turnbull, R.N., has recently communicated to the writer two cases which came on after ague at Panama—the one recovered after a year, the other was still under treatment.

To the slighter and more curable forms of Paralysis Agitans belong the *Hysterical Paralysis Agitans,* which exhibits the usual tremors (sometimes an approach to the festination), and is accompanied by hysterical symptoms and usually some disorder of the general health. Though often obstinate, it is entirely free from danger, and is usually cured when the general hysterical condition is removed by judicious treatment.

The *Intermittent Paralysis Agitans,* in which tremors of the limbs, lasting five to six minutes, recur twice or thrice in an hour, appears frequently to depend on intestinal worms in young subjects, or is a variety of the hysterical or reflex forms. It is curable.[1]

Reflex Paralysis Agitans may depend upon disordered primæ viæ, and be cured by appropriate remedies (Sauvages, Tremor a saburrâ). Perhaps derangements of other internal organs may exert a similar effect; as also external wounds and injuries. An interesting case, caused by the latter, was observed by Dr. Door, and related by Dr. Haas (1852). A healthy girl of nineteen received a splinter under the nail of her right thumb, on the extraction of which violent pain, and soon after Paralysis Agitans, came on in the right leg, subsequently spreading to the right arm and the whole body. The tongue and speech became affected; the general health suffered; the face had a stupid expression; and she dragged the legs in walking. She recovered completely.

Lastly, it appears from the important cases described by Dr.Hennis Green (a few similar to which are mentioned in older authors) that nervous tremor of the nature of Paralysis Agitans may occur *in children* (age, eleven to thirteen), and is in them speedily curable. Of the three cases which he reports, two recovered in about a month; the third died of pulmonary consumption, and no trace of lesion was found in the brain and spinal cord.

The non-senile Paralysis Agitans is particularly apt to be mistaken for chorea; it sometimes assumes the unilateral form.

2d. *Paralysis Agitans Toxica.*—Various poisons occasion debility and tremors. When these symptoms arise from the abuse of alcohol, tea, coffee, tobacco, or opium, they rarely occur except when the parts are used, and hence are simple tremors only; but if they take place also during repose, they belong to this sort of Paralysis Agitans. A strongly marked and very characteristic form of the curable Paralysis Agitans is brought on by inhaling fumes of mercury, and, though less frequently, by certain other metallic poisons (tremor metallurgorum — tremblement mercuriel). This will be described elsewhere. (See Tremblement Métallique.)

The other kinds of tremor mentioned by medical writers are either unimportant or symptomatic of other diseases; they present an interest, as related pathologically to the morbid condition probably existing in idiopathic Paralysis Agitans. Such are the tremors from bodily weakness and mental emotion: tremor senilis, which is evidently closely allied to and may pass into Paralysis Agitans senilis; febrile tremors and rigors (attended by a sense of cold, which is never present in Paralysis Agitans), analogous probably to the toxic forms of Paralysis Agitans; tremor or subsultus tendinum, which exhibits the same spasmodic jerking of the muscles as Paralysis Agitans; lastly, the tremors in diseases of the brain and spinal cord (hydrocephalus, parasites in cerebrum, myelitis, ataxia, tumours, &c.) are symptomatic, and, as previously explained, distinct from idiopathic Paralysis Agitans.

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[1] See Gowry's case.

II. *Local Paralysis Agitans* attacks a single part, most frequently the head and neck, or the arm, or the lower jaw, and remains limited to the region affected. The tremors occur occasionally, seldom constantly, during the day; they cease at night; they are excited or aggravated by exertion or emotion. Usually free from danger, local Paralysis Agitans is regarded, like the spasmodic tics, as an infirmity or bad habit rather than a disease. It is at the same time very obstinate, in fact usually incurable. It is important to distinguish the idiopathic Paralysis Agitans which continues local from that which is the precursor of the progressive general disease, or which may be symptomatic of a tumour or other lesion of the nerve centres. At first the distinction may be impossible; but the history and course of the affection determines the diagnosis. Whenever the tremor has continued for some time unattended by any concurrent serious symptoms and strictly confined to one part, experience warrants the conviction that the morbid action has been exhausted in a circumscribed area, and that no extension of the disease need be feared. It is, indeed, singular that after a few years the local exhibits no tendency to pass into the general disease.

A few remarkable cases have occurred of Paralysis Agitans restricted to the lower jaw and tongue; in some distressing instances all remedies proved unavailing. The pathology has not been ascertained, and probably the severer cases were not idiopathic, but were symptomatic of some grave disease of the nerve centres. Of the latter kind an interesting case is recorded recently by Leyden, in which Paralysis Agitans of the right arm was found associated with sarcoma in the left optic thalamus. The more serious symptomatic kinds are distinguished from the idiopathic by the more dangerous character of the symptoms, among which are the signs of centric nervous lesion, such as motor and sensory paralysis, &c. In respect of pathology and treatment, the local resembles the general Paralysis Agitans.

CAUSES.—These are frequently obscure, but it is probable that conditions productive of debility or atrophy of the motor nerve centres occasion the idiopathic Paralysis Agitans. The results of experience are as follows. *Predisposing Causes.*—1. Age is of primary importance, both in causing the disease and aggravating it. 2. Hereditary and parental influence is indicated in some cases.[1] 3. The male sex is almost exclusively the subject of general Paralysis Agitans; the hysterical forms and local tremors of the head being met with in the female. *Exciting Causes.*—1. Violent muscular exertion is a frequent cause, as also—2. Injuries, especially falls; also wounds, &c. 3. Excessive mental exertion, and particularly—4. Violent emotions, as terror or fright, which have sometimes produced the disease suddenly. 5. Venereal excesses have been alleged. 6. Exposure to cold and wet, as lying on damp ground, especially when giving rise to—7. Rheumatism, which was noted by Parkinson, and has a decided causal relation to Paralysis Agitans; also—8. Ague.[2] In certain cases Paralysis Agitans appears to have followed—9. Fever (typhoid and various exanthemata) and—10. Syphilis. 11. Intestinal worms sometimes give rise to it in young subjects, occasionally modified into a periodical or intermittent type. 12. Disordered primæ viæ (tremor a saburrâ). 13. Suppressed itch will hardly now be admitted.[3] 14. Alcohol, opium, tobacco. 15. Certain poisons, particularly mercurial vapours, cause the Paralysis Agitans toxica. While these are the causes of the idiopathic disease, the symptomatic Paralysis Agitans, as already stated, may occur, combined with other characteristic signs of nervous disease, in various lesions and tumours, &c., of the brain and spinal cord.

DIAGNOSIS.—Idiopathic Paralysis Agitans is sufficiently characterised to be of easy recognition; but its relations to allied affections are important. 1st. It is distinguished from the other species of the class tremors, because in Paralysis Agitans the trembling occurs not only during action, but even when the parts are not in use and are supported (spasmodic, tremor coactus). Tremor senilis, which most resembles it, may pass into Paralysis Agitans, when the tremors which begin during action continue after it has ceased: the tremors usually at the same time increase in intensity from trembling to jactitation. 2d. The different kinds of Paralysis Agitans are distinguished: the *simple senile* form by occurring in advanced life, by its progressive course, association with general decay of the system, and fatal issue; the *procursive* (festinans) *senile* form presents in addition the disturbed balance in locomotion; the *simple non-senile* form occurs in middle age or under, is often stationary in its progress, may be ameliorated or cured, and is not accompanied by disordered equilibrium; the *hysterical, remittent, reflex* forms, and that occurring in children, &c., are known by their special circumstances; the *toxic Paralysis Agitans* is recognised by the cause, and by the concomitant effects of the poison,—in the mercurial tremors the tongue and mouth are usually and early attacked, which is not the case in ordinary Paralysis Agitans. 3d. The relations to common chorea, both of difference and resemblance, are instructive. Chorea occurs chiefly in the young before puberty; Paralysis Agitans attacks the middle-aged, and especially the old. The gesticulations in chorea are jerking, irregular movements, changing frequently, and dissimilar on the two sides: the tremors or jactitations in Paralysis Agitans consist of to-and-fro oscillations of the part, due to the brief alternate action of antagonist muscles; they continue long unchanged, and are usually the same on the two sides. Chorea specially attacks the female, Paralysis Agitans the male sex. With attention, therefore, the diseases are not difficult to distinguish. But their points of resemblance are striking. Both consist of involuntary, spasmodic movements, rapidly repeated, and not under the control of the will, while the voluntary motor power persists in the affected parts, although it is often enfeebled, the debility sometimes amounting to paralysis.[1] Both are often caused by fright, and by rheumatism (although no relation seems to exist between Paralysis Agitans and heart disease); in both, when fatal, no visible lesion may be discovered. On the other hand, chorea is nearly always curable in a comparatively short period; while Paralysis Agitans, although susceptible of cure in younger individuals, is a peculiarly obstinate disease, and is incurable in old persons, in whom it associates itself with senile decay. 4th. The irresistible movements forward or backward in Paralysis Agitans present great affinity to the like symptoms met with in "leaping ague," and certain forms of tarantism and abnormal chorea, and which are also observed in connexion with lesion of the cerebral peduncles or other parts of the encephalon.[2] But these affections are not usually attended by tremors, and the history and special concomitant symptoms are sufficiently distinctive. The difference of Symptomatic from Idiopathic Paralysis Agitans must always be kept in view. 5th. The same remarks apply to the discrimination of idiopathic Paralysis Agitans from certain cases of locomotor ataxia and chronic myelitis, &c.—affections which it often closely resembles in the progressive character of the symptoms, and in the spasmodic nature of the movements. But these spinal diseases are, in addition to their clinical history, especially distinguished by the presence of decided motor, and mostly also of sensory paralysis; while in idiopathic Paralysis Agitans the sensibility is remarkably exempted, what is there called paralysis being only a failure of vigour. The diagnosis is very important, and only difficult because the occurrence of tremors as a complication in various organic nervous diseases may mislead, if

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[1] Sauvages: "Mulier gravida, quæ maritum subito peremptum exhorruerat, genuit filium tremore miserando correptum." Most knew a whole family in which it was hereditary. Lebert refers to females who, in successive generations, being otherwise in good health, presented tremors of the head at the climacteric age.

[2] Romberg, Maclachlan, Turnbull. No relation to gout has been alleged.

[3] Mentioned by Canstatt.

[1] Both are sometimes unilateral.

[2] Marshall Hall remarks the similarity of certain symptoms in Paralysis Agitans to the effects observed by Serres in diseases of the tuber annulare and tubercula quadrigemina. The irresistible movements—forward, backward, lateral, whirling, rolling, somersault, &c.—in experimental lesions of the brain in animals (Fodera, Magendie, Flourens, &c.) have thrown much light on the subject.

the difference of Symptomatic from Idiopathic Paralysis Agitans be not attended to. 6th. In a similar manner, Paralysis Agitans is distinguished from beriberi, raphania, &c.

COMPLICATIONS.—These are rare in idiopathic Paralysis Agitans; the health usually continuing good till senile decay begins. Apoplexy, hemi- and paraplegia sometimes occur, but not often. Common chorea has, in some cases, been found associated with Paralysis Agitans;[1] usually, however, the disordered movements called chorea have been only exaggerated examples of the shaking disease. On the other hand, as already remarked, symptomatic Paralysis Agitans may complicate many diseases of the brain and spinal cord.

PATHOLOGY AND MORBID ANATOMY.—*Tremors* are generally admitted to be a sign of weakness in the nerve centres, and are ascribed to defective and interrupted discharge of nervous stimulus. But two kinds of tremors have been distinguished:[2] *first,* simple or passive tremors, which occur during a voluntary act, and cease with it, being evidently due to want of power only (τρόμος); *second,* spasmodic, clonic, or active tremors, which take place even during rest when the parts are supported and unemployed (παλμός, tremor coactus, palpitatio): these are short, alternate, clonic convulsions of antagonist muscles, and imply some irritation in the motor nerve centres. Although these two kinds are allied and pass the one into the other, yet the distinction is important, and furnishes the ground of diagnosis; the spasmodic, not the passive tremor being characteristic of Paralysis Agitans. *The disturbance of equilibrium* is no doubt owing to an affection of a different part of the nervous system from the tremors, since these may exist, even generalized, without it. The cerebral or cerebellar peduncles, or the pons Varolii, are most probably the seat of lesion. But while the locality is different, the association with tremors shows that the morbid action is probably the same in both. The general opinion is that the tremors are due to an affection of the spinal cord, the disturbed equilibrium to an extension of the morbid action within the cranium. Hence some writers speak of *Paralysis Agitans Spinalis,* consisting of tremor and muscular debility, and *Paralysis Agitans Cerebralis,* in which the disturbance of equilibrium is superadded.[3] There is no vertigo nor distortion of the eyeballs, as in lesion of the base of the encephalon. The disturbed equilibrium seems due to weakness of one set of muscles (*e.g.* extensors), and perhaps spasmodic action of their antagonists (*i.e.* flexors).

Morbid Anatomy, which formerly gave only negative, has lately afforded indications of positive results. The facts are as follows:—

1st. In many instances of idiopathic Paralysis Agitans no lesion of the cerebro-spinal axis can be discovered by our present means of investigation. In these cases, therefore, the disease may be regarded as *functional* or *dynamical;* and it may be presumed to depend, (*a*) on impaired generation of nerve energy, due to some unknown conditions; (*b*) alteration of vascular supply, either congestion, or, as late researches on the pathology of convulsions suggest, anæmia, *i.e.* deficient or interrupted vascular supply;[1] possibly also an œdematous condition of the nerve centres might cause the symptoms; (*c*) molecular physical or chemical changes, which we may certainly assume in the toxic forms (mercurial tremors). The functional are especially the curable forms of the disease.

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[1] Maclachlan, p. 216.
[2] Distinction first clearly drawn by Sylvius, previously indicated by Galen.
[3] Remak.

2d. In more inveterate, especially senile cases, Paralysis Agitans appears to depend on a discoverable lesion; namely, an *atrophic* condition of the spinal cord, pons Varolii, crura, or medulla oblongata (*atrophic* or *organic Paralysis Agitans*). This atrophy has been found in several careful dissections, and it certainly coincides with and would explain the chief features of the disease,—namely, its obstinacy or incurability, without immediate danger to life; the progressive course; the impaired strength and muscular debility (paralysis); the occurrence in old age, after violent exertion and emotion, under conditions of premature senility, &c. In addition to simple atrophy, with serous accumulations, autopsies have revealed in different parts of the spinal cord, medulla oblongata, and pons an indurated condition (sclerosis), with patches of grey or gelatinous degeneration, due to the new formation of connective tissue, which compresses and atrophies the proper nerve structures.[2] Since a similar condition, involving extensively the posterior columns of the cord, is the chief lesion found in progressive locomotor ataxia (tabes dorsalis), some relation is established between it and Paralysis Agitans.[3] It is curious that Parkinson (from the report of a case not seen by himself) drew attention to the induration and enlargement of the upper part of the medulla spinalis, oblongata, and pons, as the probable morbid condition in Paralysis Agitans, and supposed it might be due to simple inflammation, or rheumatic or scrofulous affection of the nervous substance or membranes. The sclerotic atrophy does not seem to be of inflammatory origin, although, according to Rokitansky, it is preceded by congestion. In the early stages, there may be softening instead of induration. Rheumatic and other morbid diatheses may probably dispose to it. Degeneration of the blood-vessels may possibly be connected with it.[4] The pathogenesis of atrophy of the nerve centres, however, has not yet been fully investigated; and, although highly probable, it cannot yet be positively affirmed, that Paralysis Agitans depends upon atrophy, simple or sclerotic, of certain parts of the cerebro-spinal axis. In regard to symptomatic Paralysis Agitans, the tremors are ascribed to the atrophy of the nerve substance surrounding the tumour or other principal lesion.

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[1] Marshall Hall, Kussmaul and Tenner, Brown-Séquard, &c.

[2] Bamberger, Skoda, Oppolzer, Lebert, &c.

[3] Also with tetanus, probably chorea, and with progressive paralysis of the insane. See Rokitansky, Ueber Bindegewebs-Wucherung im Nervensysteme. Wien, 1857. Cruveilhier, &c.

[4] In Skoda's case the nerve elements were destroyed in some parts of the brain, the pons, and medulla, by embryonal connective tissue; the *vessels were obliterated;* the muscles were in a state of fatty degeneration; the neurilemma of the nerves of the upper extremity was thickened. The thickened neurilemma has been observed in other cases.

But whether the disease be functional or organic (atrophic) in its nature, it clearly affects the motor centres only, exempting the sensory and the intellectual; and, further, the morbid state of the nerve centres implies not only diminished power, but some condition of spontaneous irritation, giving rise to the spasmodic jactitations even during rest. Probably the degenerative molecular changes in the nerve structures may occasion a disturbed equilibrium and consequent irregular discharge of nerve stimulus. The morbid process is presumed to begin usually in the cervical portion of the spinal cord, since the arms are apt to be first affected, and the disease presents the bilateral type. But the occurrence of the unilateral form, as well as the fact that the limbs are much earlier affected than the trunk, shows that the possibility of the cerebral centres of motion being sometimes attacked should not be overlooked. The disturbed equilibrium probably ensues when the parts in the vicinity of the pons Varolii are involved, and the extension to the medulla oblongata is indicated by the impaired speech, deglutition, &c., which supervene in the advanced stages of the disease.

Should future researches confirm the results above indicated, there would then exist a secure basis of morbid anatomy for the distinctions which authors have indicated clinically, of idiopathic Paralysis Agitans into functional and organic: the latter with, the former without, atrophy of the nerve centres; the latter mostly incurable, the former admitting of cure.

The *Prognosis* is unfavourable, but depends upon the age of the patient and the particular form of the disease. When fully established, idiopathic Paralysis Agitans is an obstinate,[1] and in the aged, with rare exceptions, an incurable disease. But, unless mixed with signs of senile decay, Paralysis Agitans does not endanger life, and its course is often indefinitely protracted. As a rule, it is obstinate in proportion to the age of the subject, and is fatal only in the old. The distinction of curable and incurable, functional and organic (atrophic), has been already sufficiently indicated. It need only be added that mere violence of the jactitations is no evidence of incurability; slight tremors are frequently the most obstinate;[2] it is the persistence of

[1] "Morbus valde pertinax," Juncker. Comparing it with apoplexy and motor palsy, &c., he says it is inferior to them in danger to life, but equals them in its resistance to treatment.

[2] Dr. Russell Reynolds, in a letter, June 1, 1865, says: "From what I have seen of a large number of cases, I am led to believe that there is a most important difference between those cases in which there is trembling only and those in which there is clonic alternate spasm. In the latter the prognosis is very much more favourable than in the former." The age of the patient and stage of the disease being, of course, taken into account.

tremors during absence of voluntary effort, and especially during sleep, the occurrence of disturbed equilibrium, and symptoms of senile decay which are of serious import. Disturbed equilibrium is apparently incurable in itself, as well as of bad augury for the disease generally. The supervention of convulsions, apoplexy, motor or sensory paralysis, indicates more immediate danger to life.

In the Registrar-General's Reports for England and Wales, from 1855 to 1863 inclusive, 205 deaths from Paralysis Agitans are recorded, 129 being males, and 76 females; on an average about 14 males and 8 females annually. Of these 205, 189 were above 55 years of age; nearly half, viz. 91, occurring at 65 years; only 16 below 45 years, one death taking place at 20 years. It may be doubted, however, whether the cases fatal below the age of 45 were true idiopathic Paralysis Agitans; more probably they were examples of spinal or cerebral disease accompanied by tremors, *i.e.* by symptomatic Paralysis Agitans.

Of the *Modes of Termination*, the principal in the senile disease is by general decay of the system. Sometimes life is cut short by the intercurrence of the usual diseases of old age. Trousseau states that in three cases which he traced to the end the patients all died of pneumonia. The non-senile forms of Paralysis Agitans are not known to have any special mode of termination.

TREATMENT.—The modes of treatment and remedies employed are numerous, but few have been attended with success. Allowance must be made for the form of the disease; the senile being mostly incurable, the non-senile obstinate, but susceptible of relief or cure. The physician must keep in mind the propriety of abstaining from remedies in inveterate cases, after a fair trial has been given to them: a fruitless perseverance would only injure the general health and excite false hopes. At the same time, general hygienic measures are always beneficial, and, by their means alone, the symptoms may be alleviated, and life prolonged to advanced age.

The methods of treatment are:—

1. *Antiphlogistic.*—This used to be commonly practised on the supposition of the congestive or inflammatory nature of the affection of the spinal cord. In some cases, in an early stage, it seems to have done good; purgatives, indeed, in judicious moderation, are useful in all cases, and they cure the forms depending on disordered primæ viæ (tremor a saburrâ). The means employed were: Venesection general or local, purgatives, diaphoretics, mercury; blisters, setons, cauteries actual and potential, moxas, &c., to the spine; frictions, stimulant embrocations, hot baths, &c. In the majority of cases, however, this practice did no good or did positive harm. The treatment now preferred, especially in chronic cases, is

2. *Tonic,* general and nervine. Hygienic regimen, nutritious but not stimulating diet; little wine; rest or moderate exercise, light gymnastics. Excessive exertion is injurious, and many cases of simple Paralysis

Agitans are aggravated by the patients when poor being compelled to work, or, when rich, endeavouring by forced exercise to overcome the debility in which they suppose their disease consists. *Subcarbonate of iron* has been a noted remedy in consequence of Elliotson having cured cases by it in persons under 50; it failed in older patients. Quinine, zinc, arsenic, nitrate of silver, chloride of gold; *strychnine*, which has apparently cured some, and failed in other cases; *ergot*, said to have been beneficial; iodine and bromide of potassium, balsams, *oil of turpentine*, sarsaparilla, quassia, colchicum, &c.; Iceland moss; mineral-waters, sulphurous or chalybeate; sea or mountain air, the milk cure, &c.

3. *Narcotics and Calmants.—Opium, belladonna, henbane* (ʒss. of Tinct. thrice daily, used successfully in functional Paralysis Agitans by Dr. Handfield Jones); stramonium; *chloroform* stops the tremors at the time, but does not appear permanently beneficial; ether, musk, camphor; *veratrin*, externally or internally, reported successful in a case by Volz; valerian and valerianate of zinc; Calabar bean, tried without success by Dr. J. W. Ogle.

4. Baths have been much resorted to, sometimes with success, often without effect. Warm sulphur baths, especially of liver of sulphur, have been specially recommended. Simple warm baths with cold douches (Romberg). Vapour baths, Russian baths, and baths of gelatine, fir-tops, mud, even animal baths, have been used in Germany. The water cure, cold affusions; sea-bathing, which rendered one case stationary (Lebert); brine baths, ice baths, first tepid, gradually made colder. It should be remembered that some of these, especially the cold-water cure, are not free from danger, and require proper caution in old persons; sometimes they aggravate the disease.

5. *Electricity and Galvanism.*—Partially successful in previous experience, electricity was found to produce no improvement in four cases observed by Gull. The interrupted galvanic current appears also to have been of little service; but the continuous current recommended by Remak proved successful in his hands with a patient aged 60, and others were benefited by it. In a man, aged 57, the disease, well marked though recent, was cured in this manner by Dr. Russell Reynolds. After five applications of Pulvermacher's chain of 120 links, daily, for one hour, the spontaneous jactitations completely ceased; the same treatment, continued every second day, completed the cure in about a month. In other instances, this means has not produced such favourable results, but it deserves a persevering trial in all cases.

On the whole, good hygienic rules, attention to any special indications, gouty, rheumatic, &c.; regulation of the primæ viæ; the administration separately or combined of general and nervine tonics, and calmants, and the judicious use of the continuous galvanic current, are the measures chiefly to be recommended. Depletion and counter-irritation are seldom required, and would in most cases be highly injurious. Time must in all cases be allowed, for the affection is obstinate. In the confirmed senile forms, we may be satisfied with arresting or mitigating, but must not expect to cure the disease.

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WRITERS' CRAMP.

By J. Russell Reynolds, M.D. F.R.S.

Definition.—A chronic disease, characterised by the occurrence of spasm when the attempt is made to execute special and complicated movements, the result of previous education; such spasm not following muscular actions of the affected part when these special movements are not required.

The term "Writers' Cramp" is bad in one respect, because the symptoms it denotes do not belong exclusively to the act of writing: it is good, and therefore retained in this "System of Medicine," because it points to the most frequent form in which the disease is exhibited, and because it has already passed into general usage.

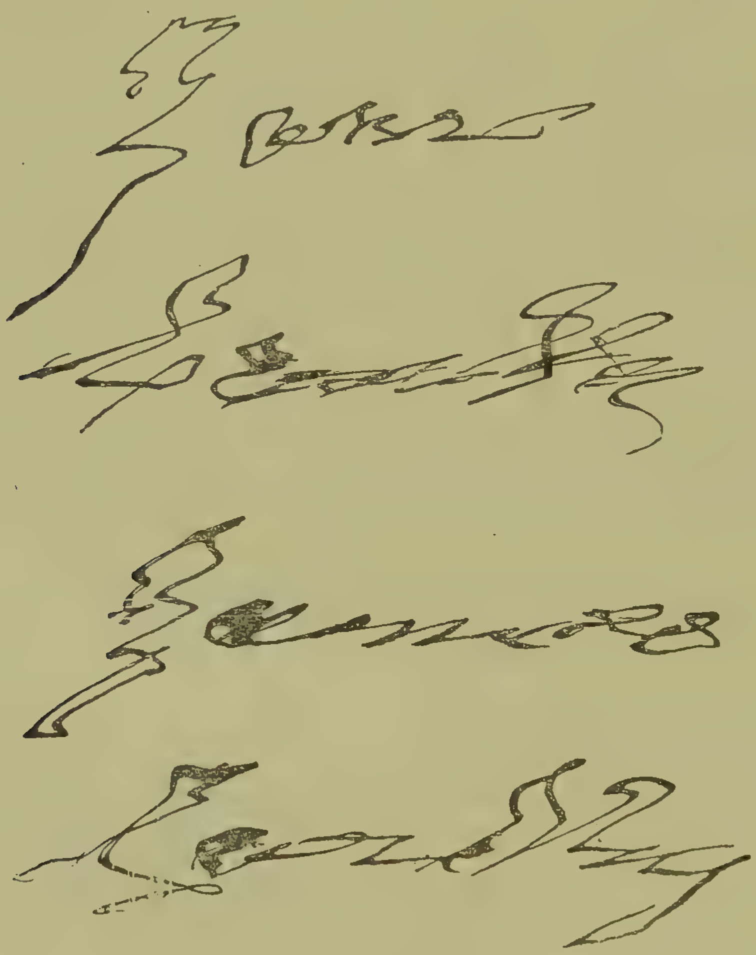
A disease pathologically similar to Writers' Cramp may be found in the artist, and may prevent him from painting in oils; it may occur in the violinist or the pianist, and hinder the musical performances of either; it may be met with in the seamstress, or the smith, or the milkmaid, and may limit or destroy their powers of work. Wherever it is found it shows the same general features, expressed in the definition, viz. a limitation by spasm of a particular kind of movement, and of that movement only.

Synonyms.—Scriveners' Palsy; Mogigraphie; Schreibekrampf; Crampe des Ecrivains; Schusterkrampf; Melkerkrampf.

Symptoms.—A slowly developed difficulty in executing a particular movement, such as that of writing, or playing on a musical instrument, other movements of the same limb being perfectly easy of performance. Usually the patient feels at first some undue weariness after long exertion, a stiffness of the fingers, or an unsteadiness and uncertainty of movement, all of which immediately disappear on giving up the exertion. If writing, a man feels that his pen does not do what he intended that it should; that his handwriting looks unnatural; that he has to hold his pen more tightly than before, in order to keep it between his thumb and fingers; that it starts from its place, and often is pushed, by his first finger, over the nail of his thumb, and that he has some difficulty in getting it back to its place. A pianist makes blunders in striking chords, the fingers falling on keys they were intended to avoid: the violinist cannot control the movements of his left hand,—or can do so only by a painful effort,—the fingers running together and feeling stiff: the seamstress cannot ply her needle, but pricks her fingers, and makes her stitching irregular. In one case, under my own care, the bricklayer could not use his trowel.

At first the difficulty is slight, and may be overcome by strenuous effort; but, after a little time, if the attempt to continue or repeat the movement be persisted in, there is distinct cramp, jactitation, or tremor of the hand, and the particular performance is quite impossible. Other things may be done, but that one thing with regard to which the difficulty was first felt cannot be effected properly by any amount of exertion. A patient may not be able to write, and yet may feel no difficulty in fingering either the piano-forte or the harp.

The accompanying woodcut represents four different attempts made by one of my patients to write his name—



The moment that the attempt is given up the patient feels nothing abnormal; but the moment that he tries again to perform this special act the difficulty returns, and increases. Sometimes the special symptoms are made worse by any exertion of the arm.

The cramp-movements, at first limited to the thumb and fingers, are sometimes avoided by the writer who adopts mechanical devices which leave them at rest, but make it possible to perform the act of writing by using only the muscles of the wrist and fore-arm; as soon, however, as he has trained himself to write in this awkward manner, the muscles of the fore-arm take on a spasmodic movement, and he is no better off than before. In one case, which I have recently seen, the patient could manage to write a few words by moving only the muscles of his arm and trunk—his pen was directed by the muscles of his back and arm, the latter being pressed closely against his side; but, after a few seconds, spasm occurred in these, the whole body was contracted, the head being drawn downwards to the right shoulder, and the trunk contorted so as to render it concave on the right side.

In several cases that I have known the sufferers have taught themselves to write with their left hands, to do this with ease, rapidity, and neatness; but, shortly after having acquired the art, the left hand has become affected in a similar manner, and its writing-power has been limited more rapidly than was that of the other limb.

When the disease has existed for some time, the attempt to write often becomes painful; there is a feeling of "cramp," and much general distress, accompanied by spasmodic movements in the neck, and sometimes in the limbs not especially engaged in the effort; and yet, apart from the attempt to write, there is no spasm and no inconvenience.

After long persistence of the cramp there is sometimes feebleness in the general movements of the limb,—the grasp is not so firm as it used to be; but such quasi-paralysis is the exception, and not the rule.

In some cases the spasmodic movements have not been so closely limited, as they are in the majority, to the attempted performance of a special act; those movements which require no fine adjustment may be performed with force and propriety, but others—needing delicate co-ordination—may be difficult or even impossible. There has been tremor, or choreiform agitation of the limb, more or less persistent during the day, even when no voluntary effort is being made, but ceasing at night, during sleep, or after prolonged rest in one position.

There are—in some individuals, but not in all—abnormal sensations in the affected limb; and these may be noticed before any cramp appears. They may be increased by exertion, but do not entirely depend upon it. They are vague in character, such as a "feeling of weight," or of "tightness," "numbness," or "coldness;" a pain, but more often a "something not quite pain going up from the hand to the back." In some cases there has been actual anæsthesia of the fingers, and an "aching in the spine."[1] There is nothing peculiar to the disease now mentioned in the sensations that I have heard described, when description was possible, except this, that the attempt to control the spasm augmented the distressing or uneasy feelings.

In the majority of cases the special cramp exists by itself; but in a few it is associated with other disturbances of the nervous system. Those which I have met with have been torticollis, occasional strabismus, stammering, and palpitation of the heart, with some distress about the cardiac region, over and above the mere fact of increased force or frequency of beat.

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[1] Solly, on Scriveners' Palsy; Lancet, Jan. 28, 1865.

The general health in some of the most typical cases has been excellent, and the physical strength equal to and even beyond the average. In a few individuals there have been weakness, a "nervous temperament," and some anæmia with impaired digestion and nutrition; but in no one has there been witnessed any modification which is not consistent with, and frequently encountered in, other diseases.

ETIOLOGY.—*Age.* Early life appears to be exempt; I have not met with a single case in which the symptoms appeared before the age of thirty. *Sex.* The male sex is much more liable to suffer than the female. *Occupation.* It is commonly held that the disease is caused by excessive exertion, but there are reasons for doubting the correctness of this statement. Thousands of individuals write, work, milk, or play musical instruments to the highest degree that is possible, without suffering from the least inconvenience of the kind now described; and, on the other hand, many cases occur in which there has been no excessive strain upon the muscles in the performance of these special acts; and, indeed, in some quite characteristic examples of the malady, there has been less than the usual amount of writing performed by gentlemen of the age and professions of my patients. It may then be convenient, but it is not scientific, to refer this form of cramp to over-exertion of a special kind.

Worry of mind and anxiety have been present in many cases before the outbreak of the symptoms, but so they have been in many other forms of nervous disturbance quite different from this; and in some persons affected with Writers' Cramp there has been nothing of the kind to which the patient or others could refer the symptoms. I have known the symptoms of Writers' Cramp to occur in one who had been much interested in their appearance in a friend. An *injury to the arm* has been supposed, in some cases, to have originated the disease.

DIAGNOSIS.—Scarcely anything need be added to the description already given. The special character of the difficulty is the diagnostic mark of true Writers' Cramp. A man may be unable to write from *lead poisoning*. But the presence of paralysis rather than spasm; the singling out of certain muscles not only for weakness, but for loss of nutrition and of irritability to electricity in the induced form; the equal affection of the two upper extremities, although, when slight, it may be shown more conspicuously in the hand which writes, and has been educated to perform other complex movements; the presence of a blue line on the gums, and the general history of saturnine intoxication,—are sufficient to establish the diagnosis.

Wasting Palsy, which often commences in the muscles of the thumb, may be known by its characteristic feature, "wasting," and needs only to be mentioned in order to be distinguished from Writers' Cramp. In wasting palsy the loss of power is in direct proportion to the loss of nutrition; in Writers' Cramp, it is the spasm which interferes with the particular movement that is required.

Local Paralysis.—A few weeks ago a gentleman was sent to me with supposed "Scriveners' Palsy." He had been reading and writing much, and on one evening sat reading for some hours "in a draught;" his hand was weak, and on the following day he could not write. There was when I saw him nearly complete paralysis of the right hand and fore-arm, and the electric irritability was almost extinct, but in the course of a fortnight the power had returned, and the patient was well. The extent of the paralysis and the absence of spasm were the distinctive marks. Several cases of this kind have come before me, and have been thought to be examples of Writers' Cramp; but the fact of their having been mistaken for the latter is enough to put any one on his guard against a repetition of the error.

PROGNOSIS.—If the case be seen when the symptoms have existed for only a short time, relief may be confidently expected, provided that rest can be given. If the symptoms have existed for many months, or if rest be impossible, the prognosis is extremely unfavourable. There is scarcely any malady which has resisted more obstinately all kinds of attempts—well-directed and ill-directed—which have been made for its cure. Many who were seriously threatened with Writers' Cramp are now free from the malady because they rested; many who could not and did not rest, are now, in the present state of therapeutics, incurable.

Bearing in mind what has been said with regard to the extension of the disease into other regions of the nervous system, some caution is required in stating the general prognosis of such cases, but, in the vast majority, it may be confidently expected that no such extension will occur. When there are signs of disease already present in other directions, such as strabismus, torticollis, weakness of the corresponding leg, and the like, the prognosis should be extremely guarded.

PATHOLOGY.—The exact locality of disease, and the precise nature of the change which constitutes it, have not been yet demonstrated with regard to Writers' Cramp. Its closest clinical affinities are with stammering, spasmodic wry-neck, and histrionic spasm, or "muscular tic" of the face. Analogous maladies, but moving in a yet wider range, are sometimes encountered, such as certain forms of rotatory movement, of chorea, and of locomotor ataxy; and beyond these there are anomalous cases, which every physician occasionally meets with, but does not know how to designate. One patient cannot make the attempt to walk without performing, or running in danger of performing, sundry rotatory movements, which terminate in a fall;

another, a hard-working clergyman, can only speak, though he has the voice of a Stentor, when on a level with his audience; and this not from any fear, or shyness, or sham, but from definite aphonia. Some of the spasmodic movements induced in frogs by injury to the auditory nerve are of similar character;[1] and the experiments of Magendie, Flourens, Longet, and Schiff afford further illustrations of analogous disturbances in the physiology of motion.

In order to understand Writers' Cramp, it is necessary to remember what is, physiologically, involved in the education of the muscles to perform complicated acts, such as those of writing, speaking, or playing on musical instruments. The will does not pick out the muscles which are to be brought into play to hold a pen; it simply directs itself to the result. The boy who plays at marbles directs his movements in the same manner, and with as much accuracy and nicety, as the professor of anatomy directs his when he is writing a description of the muscles of the hand. The combination or co-ordination of muscular contractions is determined by the will, but is effected by another agency. Each is conscious of a wish to do a certain thing, and of a will to do it, but a knowledge of the mode in which the movement is brought about does not help, and may sometimes hinder, its production. Experimental physiology and clinical pathology combine to teach us that a certain portion of the nervous system, the cerebellum, has the power of effecting the co-ordination that is required; and they also unite in proving—what is often lost sight of—that this co-ordinating faculty is guided by sensations, and can act efficiently only when they are normal in kind and intensity. The production of a movement such as writing is therefore a very complicated process, requiring for its efficient performance the integrity of a great number of different parts: viz., that of the will and its immediate exponent in the cerebral hemispheres; that of the nerve-fibres between it and the muscles, together with that of the ganglia which exist on certain nerve-trunks; that of the muscles themselves; that of the cerebellum, as the centre of co-ordination, also that of all the "sensory" nerve-fibres which place it in relation with the organs of special sense and with the muscles themselves; and, lastly, integrity of the organs of sense, so that they, at the peripheral expansion of their special nerves, can receive impressions in a normal manner. It must be remembered, also, that not only the fact but the degree of contraction is under the control and guidance of the same organs or parts of organs. Failure in any one portion of this apparatus interferes with the production of the movement that is required; and the kind of failure is determined by the locality of the lesion; or, in other words, by the nature of the process or function which is lost or disturbed. If the contraction of a muscle be acutely painful, the man cannot write, the act would be impossible in some cases of rheumatism; if the muscle be wasted, it cannot be put into the same amount of contraction as in health; if the skin have lost its sensitiveness, all fine movements are awkwardly performed, and the finest are rendered impossible; they may be partially guided by the eye, but the guidance is defective for the most complicated acts; if the motor nerve be damaged, the muscle is *pro tanto* palsied; if the sensory nerves be injured, sensation is defective; if the sense of muscular condition be in abeyance, the power to control either the kind or force of contraction is without its guide: but locally, *i.e.* so far as that limb is concerned, all other nerve, muscular, and sentient properties may be intact, and yet spasm or paralysis, or both, are present. If the will be deficient, and this from any cause, there is palsy, or irregular movement; if the fibres coming between it and the nerve-trunks be injured, there is paralysis in the ordinary sense of the word; if the cerebellum be diseased, there is loss of co-ordination, while power and sensation persist; if the spinal cord be injured, there may be, in relation to the nature and locality of the injury, almost any one of the conditions that have been enumerated.

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[1] Brown-Séquard, Lectures on the Physiology and Pathology of the Central Nervous tem, p. 194.

In true Writers' Cramp, the will, the co-ordinating power in all directions but in one, the motor power, the muscular nutrition and activity, as well as the sensorial faculties, are uninjured; the individual is, or may be, "well" in all respects but one. A particular kind of movement is interfered with, by the occurrence of irregular and spasmodic, instead of regular and co-ordinated, contractions. It must be carefully remembered that the malady is special; the muscles which cannot be made to write can be controlled so as to fasten the most tiresome buttons, carve the toughest of pheasants, or pull a heavy boat. The pianist cannot play on the pianoforte, but he can write as well as ever; the bricklayer cannot use his trowel, but he can do everything else that he wants to do; and in order to understand this, we must revert to what is included in education, and what confers the dexterity which comes of special practice. Many movements are "automatic;" we adopt them without education and without effort; others are the result of laborious "practice." It would seem that the body is naturally endowed with certain paths or lines of nerve-action, along which all moves easily. The instinctive movements of the child or of the animal are examples of the mode in which, along these lines, impressions from without pass readily, and become converted into motor impulses, which are, in their turn, conveyed to muscles, which contract, and so perform these instinctive acts. But the process of education, so far as the performance of writing, playing, stitching, &c. is concerned, consists in the frequent repetition, by an act of the will, of certain forced and complicated movements. The repetition makes them easy, until at length they are executed without effort, and almost unconsciously. It would seem that, by this education, new paths are forced, so that what was once difficult and required attention becomes day by day more easy, and at last "secondarily automatic." It cannot be doubted that some changes take place in the nutrition of the parts through which these lines of nerve-action run; and that their education involves structural alteration in the organs. The perfection with which complicated movements are performed in the lower animals appears to be associated with great keenness and remarkable development of the organs of sense; and in man a similar relation may be observed. No man writes well who has not keen sight and a quick sense of touch; no man plays well on the violin who has not an acute ear, and a delicate power of feeling in his fingers. In all instances of educated movement some "sense" is needed, and is an important element in the process by which the result is obtained.

In the present state of science it is not possible to say, for every act, what part of the nervous system is especially engaged in this educational development; but it seems probable that the association of movement is effected by ganglia which are common to fibres passing through distinct but contiguous nerve-trunks, and that it is owing to some nutrition-change in them—the result of persevering and forced effort—that the perfection of movement is produced; associations at first caused by the will, are at last produced unconsciously. What happens, then, in such maladies as Writers' Cramp, is a perverted nutrition of these parts; a worn-out activity, or a degeneration which may arise without over-exertion, and destroy all that had been previously achieved.[1] Neuromata have existed in the arms of some patients. The disease, as it has been shown, passes readily from one side of the body to the other; and it must be carefully borne in mind that co-ordination of movement is a most complex process, requiring integrity of sensation as well as of motor nerve and of cerebellum. The real mischief may be some want of limiting and guiding influence ordinarily coming, through sensation, from external impressions. The spasm which occurs is very like that which Mr. Lockhart Clarke describes as taking place in the legs of ataxic patients who cannot regulate the *force* of their muscular contractions.

TREATMENT.—In an early stage absolute rest may do much; in a later stage it may accomplish something; but I know of nothing else which can be called a therapeutic agent. I have tried every form of general and nervine tonic, of sedative, and of local application, but no one of them has been of the least specific value. I have used hypodermic injections of morphia, atropine, and of arsenic, and have found them incompetent to cure the disease. The hypodermic injection of morphia appears sometimes to relieve the spasm for a certain period, and I have seen the writing become steadier within five minutes of the application, and increase in precision for half an hour, but the effect has then, or soon afterwards, passed away, and a frequent repetition of the process has been without any permanent result. It has, unfortunately, happened that several patients in whom I have used morphia hypodermically presented an intolerance of that medicine. I have employed galvanism and electricity in all their forms, and have seen no good result. But in many cases perfect rest has removed the symptoms, and it alone seems worthy of being regarded as a means of cure.

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[1] Some of these points in the pathology of Writers' Cramp have been ably treated by Mr. Solly, in the Lectures already referred to in the Lancet of 1865.

Mechanical contrivances for holding the pen may render occasional writing possible, but they do not affect the disease; and persistence in their use has been followed by an extension of the malady to the muscles of the fore-arm and arm.

CONVULSIONS.

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P.

IT cannot be kept too much in mind that Convulsion is a symptom, not a disease. But it is the most striking member of the series of symptoms in which it occurs, and in many cases the only one about which we have definite knowledge. In other words, although we always believe a Convulsion to be symptomatic, we too often know very little of the condition of the system of which it is one of the symptoms; and this even after post-mortem examinations. Let us glance at the circumstances with which Convulsions may occur.

Convulsions occur in association with organic changes in the nervous system of the most varied kinds; for instance, with cerebral hæmorrhage, and with intracranial tumours. They follow injuries to the head, either immediately or remotely: immediately (within a few hours), as when a blow leads to meningeal hæmorrhage; and remotely (after weeks or months), when diseased bone, the consequence of a blow, causes cerebral abscess. They will occur in a healthy but parturient woman after severe loss of blood. They occur with diseased kidney. They come on as indirect results of syphilis, as in cases of gummatous tumours in the hemisphere. In children they are often associated with rickets. Some believe that Convulsions may be the results of disturbances of parts of the body at a distance from the central nervous system, the result of eccentric irritations, such as the irritation occurring with dentition, or the irritation of worms. Finally, there are a large number of cases of convulsive seizures which (for want even of that approximative knowledge we have of such causes of fits as are mentioned above) we are obliged to speak of as essential, eclamptic, epileptic, or epileptiform. When we consider further that the symptom occurs at all ages and in many diseases, that there are many varieties in parts of the body affected by spasm—it is unilateral or general—many degrees in severity—there may be local spasm without loss of consciousness, or general convulsion with profound coma—and in times of recurrence—there may be one fit a week, or fifty in a day—we are forced to the conclusion that we can only speak of Convulsion as a symptom.

The only things we can safely affirm of the symptom are certain truisms. It is the phenomenon of an occasional discharge of nerve tissue (no doubt of grey matter). It points not to destroying lesions, but to unstable nerve tissue—to "functional" changes. But the most careful study of the symptom (the paroxysm) tells us nothing of the *pathological* process by which such changes of instability are brought about; does not, for instance, enable us to say whether these changes are the result of "irritation," of tumours, of uræmia, or whether they are not minute changes (epileptic) to the pathology of which we have no clue. But clinical study of the *circumstances under which the symptom occurs,* tells us very much. Although it rarely leads us to a knowledge of the pathological condition of the nervous centres, it gives valuable information as to the treatment of the patient, and for the purposes of prognosis. There is a practical convenience in studying this symptom separately. Indeed, we are forced to this narrow study, as it is very often our only "way in" to a case; and its distinct consideration will not be hurtful if we use it as a point about which to group not only our positive knowledge for present action, but also, if such a phrase may be permitted, our positive ignorance for future research.

I will try to show what meaning we can give to this symptom under various circumstances; how we should investigate the condition of our patient who presents it, and what we should try to do for him. It may be well to say that I have only to consider epilepsy so far as diagnosis is concerned. It is justifiable to sacrifice some exactness to convenience by dividing the subject into (1) Convulsions in infants and young children, and (2) Convulsions in persons above seven years of age.

CONVULSIONS IN CHILDREN.

Convulsions may occur at any age, and this remark applies not only to general Convulsions but also to most kinds of convulsive movements. Still, Convulsion is *par excellence* the nervous symptom of infants and young children.[1] The tendency to Convulsions gradually decreases with increasing years. The following quotation from West shows this:—"In proportion as the brain increases in size, and its structure acquires perfection, and its higher functions become displayed, Convulsions grow less and less frequent until, from the tenth to the fifteenth year, they cause less than three per cent., and above fifteen less than one per cent. of the deaths from diseases of the nervous system." The first line in the accompanying table (Dr. West adds in a footnote) shows the proportion per cent. of deaths from diseases of the nervous system at different ages, to the deaths from all causes at the same ages in the metropolis; and the second line the proportion borne by deaths from Convulsions, to deaths from diseases of the nervous system in general:—

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[1] Meigs and Pepper write:—"During the five years from 1844 to 1848 inclusive, 1,729 children under fifteen years of age died in this city (Philadelphia) of convulsions, whilst, during the same time, 1,611 died of infantile cholera, 1,060 of marasmus, 1,041 of dropsy of the brain, and 772 of pneumonia, showing that eclampsia was the cause of a larger number of deaths than any other of the diseases just mentioned. It must be recollected, however, that a very large number of these cases ought, beyond doubt, to have been returned under other titles, as many of them must have been a mere result of organic disease of the cerebro-spinal axis, and of other acute local or general diseases."

Under 1 Year.	From 1 to 3 Years.	From 3 to 5 Years.	Total under 5 Years.	From 5 to 10 Years.	From 10 to 15 Years.	Total above 15 Years.
30·5	18·5	17·6	24·3	15·1	10·6	10·4
73·3	24·9	17·8	54·3	9·9	2·4	·8

West says: "In a large proportion of cases of Convulsion in the infant, Convulsions answer to delirium in the adult;" and Trousseau says that there are children who have Convulsions as easily as some have delirium or even dreams.

We shall then, as a preliminary, speak briefly of the *physiological* peculiarities of the child's nervous system. We may affirm of it two things. (1) It is undeveloped. Besides the obvious fact that the infant has to acquire such movements as those of walking and talking, there is evidence from the special nervous diseases of children, that the parts of the young nervous system are not knit together so closely as in the adult—incomplete neurification analogous to incomplete ossification. The child is the subject of certain *limited* palsies and limited spasms which do not occur in the adult. Indeed, there is a form of talipes varus, Dr. Little tells us, which is always congenital. "Infantile paralysis" never occurs in adults. After hemiplegia in childhood, a well-known contraction (spastic rigidity) often sets in which does not follow hemiplegia in adults (p. 260). And lastly, coming near to our immediate topic, spasm of the glottis is a convulsion of a certain limited region which is rarely met with after the age of three or four years. Then there are minor symptoms which are almost peculiar to children, *e.g.* carpo-pædal contractions. As regards the last two symptoms, there are the significant exceptions that they occur in hysterical women.

(2.) The nervous system is developing. It is in a state of active change. Its nutrition will be in considerable excess of its expenditure, whilst in adults the two will be more evenly balanced. For this reason the child's nervous tissue will naturally be more unstable than is that of the adult. It will more easily discharge from a slight cause, or, to use a common expression, it is more excitable. It is believed too that the equilibrium of the child's nervous system is more often upset by *nerve-transmitted irritations* than that of the adult is. Hence very severe convulsions are ascribed to irritation carried by the fifth nerve from the gums during dentition, or to the irritation of worms. The child's nervous system is even believed by some to be naturally so unstable that eccentric irritations so very local as those just mentioned will produce a general convulsion in a *healthy child,* i.e. will determine a sudden and excessive discharge of nervous tissue which is only *physiologically* unstable. Others will qualify this opinion by the supposition that the nervous system, or some part of it, is *pathologically* unstable prior to the action on it of the transmitted irritation. Thus the late Dr. Hillier says (I italicise some words): "It is very doubtful whether in a *healthy* child these causes can produce convulsions at all; in a *predisposed* subject they no doubt often *excite* them."

The above are physiological differences. Disease finds the child's nervous system undeveloped, and it finds it developing. But, so to speak, the attacking disease itself has peculiarities, at all events negative peculiarities. A child is much less likely to suffer from gross lesions in the brain, such as hæmorrhage, syphiloma, and other new growths; he is less likely to suffer from uræmia excepting from acute changes of the kidney, especially of scarlatinal origin; or, putting it more simply, we usually *discover* no pathological changes in the nervous system of a child who has died of Convulsion. Wilks, speaking of diseases of children, says: "We meet with a large number of cases where the post-mortem appearances are absolutely nothing; and where, indeed, we could scarcely expect to find it otherwise. We allude especially to cases of *Convulsions* in children where no morbid changes are discovered; and when we consider that a child may have several convulsive attacks and speedily recover, which only a degree more severe shall prove fatal, it is clear that no very great change could occur in an organ which would have perfectly recovered itself had the fit been only one degree less in severity." Whilst it is true that, as a rule, no pathological changes are discoverable, we must not infer that pathological changes do not exist: the probability is that there are minute changes. Nor must we infer from complete recovery from a convulsion, or a series of convulsions, that there are no pathological changes. Adults recover even from hemiplegia, which subsequent post-mortem examination shows to have been due to obvious although very limited destroying lesions—small clots, for instance. Therefore recovery from Convulsions is no certain sign that there was no real impairment of structure. It is a sign only that no wide breaking up of structure has happened. There must certainly be local changes in those cases of Convulsions in which hemiplegia follows, however temporary it may be, since local symptoms of necessity imply local lesions. Still this is only a necessary inference, as we rarely *discover* any changes even in these cases. Nay, even in those rare cases where we find *gross disease*, a tumour for instance, we do not discover the *minute* and *secondary* changes on which the discharge producing the Convulsion depends. We must not say that the tumour was the direct "cause" of the Convulsion, but that it led to secondary changes in nervous tissue on which the Convulsion depended, and these secondary changes are inferred, not demonstrated.

It is true that there are found at examinations after death from Convulsion abnormal quantities of serum and blood in the head, but these differences are quite as likely to be results of the fits as their causes—the results of the sudden interference with respiration.

Effusion of serum and congestion of the brain have not been shown, either in adults or in children, to have much to do in producing sudden and severe cerebral symptoms of any kind. Of course those cases in which, possibly from obstruction to the vein of Galen, as by tumours of the vermiform process of the cerebellum, there is immense effusion into the cerebral ventricles, are not in question.

To resume, we know nothing of the causes of Convulsions in children in the sense of knowing what the pathological changes are. This is so, however much we may narrow our consideration to groups of cases, either to those which occur singly and at intervals over a period of months or years, and which are often called epileptic, or to those in which the fits occur in considerable number for a limited period, and are often called eclamptic.

After these general remarks on the symptom Convulsion as it occurs in children, we have to consider what meaning we can give it in particular cases. Our task is twofold. We have first to note carefully the *kind* of *paroxysm*. For instance, is the Convulsion one-sided? Is it followed by hemiplegia? Secondly, to investigate the child's *general bodily health*. For instance, is he rickety? Is there bronchitis? Is there irritation from dentition?

PAROXYSM.

Convulsions occur in all degrees. The Convulsion may be a twitching or clenching of the hands only, or an occasional grim smile in sleep, or the spasm may be general and so severe that the child dies of the paroxysm, even of the first. Attacks of slight occasional spasm, be it of one finger, have the same *general* significance as a severe convulsion has. They are both Convulsions, the proof being that, as in adults, we have very often first the local and quasi-trivial spasm, and later, a general convulsion. Each of them is a sign that there is an abnormal discharge of the nervous system or of some part of it. But the *effect* of the severe and of the slight discharges is different. We have to consider very carefully not only the "cause" of the Convulsion, but also what effect single and repeated paroxysms produce on the child. The slight and partial fits do no harm, or little harm, for they do not spread to the respiratory muscles, and thus, as it were, they do not retaliate on the nervous system which "began it" by congestion of the brain, and they are too slight to exhaust the child by abnormal exercise of the muscles convulsed. Further, it is usually held that fits so slight and so partial point to slight and usually to transient causes, and that they often disappear when we obviate some condition of ill-health, such as wrong feeding and diarrhœa, or when we lance gums swollen during the eruption of teeth. But even granting that these slight symptoms signify that the nervous system is but slightly disturbed, or that they usually directly result from some removable condition, and that the seizure does little harm to the patient, they still demand serious consideration, for two reasons. In the first place, we think ill of that nervous system which is upset ever so little by slight causes, such as over-eating, and we have anxieties that if the child be afterwards exposed to severer exciting causes, such as fright, exhausting diarrhœa, &c., very severe convulsions may occur. Another reason for careful attention to slight spasms is, that they may not disappear under treatment, or that they disappear for a time only, and that they are often the premonitory symptoms of severer convulsions, the paroxysms of which *will* do much harm to the child by interfering with respiration and, when frequently repeated, by exhausting him. Therefore, although in strictness these slight symptoms are themselves miniature convulsions, it is convenient to consider them, along with other symptoms, as warnings. But it must be remarked that, like an adult, a child may be suddenly attacked by a severe convulsion in the midst of what seems to be perfect health. He may die in the first fit, or we may see him soon after the seizure playing about as if nothing unusual had happened.

PREMONITORY SYMPTOMS.—It is proper to mention, to begin with, that Trousseau states that there are no premonitory symptoms. "Nothing," he says, "foretells the invasion of the attack; and, for my part, I have never observed the premonitory signs spoken of by Brachet, and repeated after him by others." Most authors, however, admit that there usually are warning symptoms. There are often symptoms before there is any local twitching, such as peevishness, want of sleep, and sleepiness. These symptoms show that the child's nervous system is suffering, but they cannot, of course, be taken as evidence that the illness is one in which the symptom Convulsion will be the sole or even the most striking event. In the child, as in the adult, want of sleep is associated with drowsiness. Adult patients will sometimes say what very young patients are not likely to tell us, that they always feel sleepy and never sleep soundly. Children who are about to have Convulsions will sleep with their eyes partly open; their mouths will twitch; they will start in their sleep, grind their teeth, and may have night terrors. In the day they are dull, heavy, and peevish. When any twitching occurs on *one* side of the face or in one limb, or in both limbs of *one* side, however slight the cause, let us say over-eating, which seems to excite it, we fear the nervous system is seriously implicated, and that severe convulsions are setting in. When the slight symptoms occur during waking, especially if now and then a vacant look points to some loss of consciousness, however transient it may be, we fear severe convulsions are at hand. I may here quote, as a summary of the occasionally insidious march of the symptoms, what Churchill says of fits of dentition. "I have frequently observed a sort of gradation from simple irritation and restlessness to starting, surprise, wildness of look, partial or local convulsive movements, and, lastly, general convulsions."

Having spoken of partial or slight fits and incidentally of premonitory symptoms, we now come to consider varieties of severer convulsions. It is not denied that we may have any kind of occasional spasms in children, but we choose three types. It is a very important matter to note the variety of Convulsions especially for prognosis:—1. Laryngismus Stridulus; 2. Unilateral Convulsions; 3. General Convulsions.

Laryngismus Stridulus.—A certain kind of Convulsion is called laryngismus stridulus because the muscles of the larynx being attacked by spasm, a noise results during inspiration from narrowing of the glottis. It is not a laryngeal disease, although one of its names, "false croup," seems to imply that it is. There is no continued fever in laryngismus. It is Convulsion affecting the muscles of respiration. Dr. Gee, in a most able article, very rich in clinical observations (Convulsions in Children, St. Bartholomew's Hospital Reports, vol. iii.), remarks: "In laryngismus (convulsion interne) we have a disease closely allied to epileptiform convulsion. Out of fifty cases of laryngismus of which I have notes, nineteen had had eclamptic fits." Niemeyer treats of it under the head of Nervous Diseases of the Larynx (Spasm of the Muscles of the Glottis). The larynx is found to be quite normal post-mortem. The obtrusive symptom, the crowing noise, is due to spasm of the glottis, but in many cases the muscles of the chest and abdomen, as well as those of the larynx, become involved.

The alliance of the laryngeal spasm—the local convulsion—with general convulsion is further shown by the fact that not infrequently the child has first laryngismus and then general convulsion. Occasionally we find in the intervals of the fits tonic spasm of the hands and feet—carpo-pedal contractions.

Yet it has certain peculiarities beyond those of limitation of range of the spasm. Age is one of the most important of these. Dr. West compares attacks of laryngismus to hysterical attacks, and remarks that both occur when processes of development are active. Out of thirty-seven cases of laryngismus, thirty-one, he tells us, occurred betwixt the ages of six months and two years. Vogel[1] says, "The age at which the disease occurs, fluctuates between one-half and three years; that is to say, it makes its appearance with the eruption of the first tooth, and disappears with that of the last." Niemeyer says that spasm of the glottis occurs almost exclusively during childhood, and especially in the first year of life. It is most frequent during the period of the first dentition. He makes a remark which is of considerable interest in connexion with the one quoted from West: "Among adults none but hysterical persons suffer from spasm of the glottis, and those only exceptionally." Mackenzie, in his work, "Nervo-muscular Affections of the Larynx," treats of laryngismus

[1] A Practical Treatise on Diseases of Children. By Alfred Vogel, M.D. (Translated by H. Raphael, M.D.) Appleton & Co., New York.

under the head of Spasm of the Adductors of the Vocal Cords, and says that hysterical cases (in adults) are by no means infrequent. The observation of these cases is of very great interest, because in the adult the condition of the vocal cords in the attack can be seen, and we may plausibly infer that the condition of the glottis is similar in the laryngismus of children. Mackenzie writes of adults: "With the laryngoscope the vocal cords can be seen on inspiration to be spasmodically approximated. They may separate widely; but, instead of remaining apart for a few seconds, they are instantly and spasmodically adducted to the median line, or even beyond it, that is, against one another." Another peculiarity is, that boys are much oftener the subjects of this disease than girls, "a fact almost all authors admit" (Vogel). Of Gee's (op. cit.) forty-eight cases, thirty-four were males. Mackenzie (op. cit.) says, "The greater liability of the male sex, which occurs in other laryngeal diseases, holds good here."

The most striking feature of the disease is the crowing noise the child makes. This noise often begins insidiously, but there is great difference in this respect. The crowing noise is most frequently observed when the child awakes from sleep, and is very often noticed for the first time in the night. After several attacks of the crowing, which is generally at first occasional, and produces little inconvenience, very often exciting no alarm in the child's friends, a severe paroxysm may come on. A slight crowing noise may become almost continuous in the child's ordinary respiratory movements, and a severe attack may come on in the midst of this warning. The convulsion may be, it is believed, at first no more than spasm of the laryngeal muscles—a laryngeal convulsion; but in many cases the whole system of respiration is involved in the spasm, and sometimes the limbs—the convulsion becomes general. The severe attack is paroxysmal, and while the paroxysm is on, respiration is much impeded—sometimes indeed being quite, for a short time, suspended, as in severe convulsion in the adult. At the climax the face is flushed, the eyeballs start, the veins of the neck are distended, and the face wears an aspect of exquisite distress. The sign that this stage of danger is passing is a crowing or whistling noise made by air entering the now only narrowed glottic aperture. As before said, general convulsion may supervene. In the intervals, as in the other forms of Convulsion, if the attacks be not frequent, the child may be quite well, or only fatigued and peevish. If they are frequent (and they may occur thirty times a day), the child may be exceedingly exhausted, almost comatose. Occasionally, although this is a rare occurrence, the child may die in an attack, even in the first attack, just as now and then an adult may die in an epileptic attack. Nay, according to Niemeyer, in rare cases spasm of the glottis in hysterical adults produces death by suffocation.

Without underrating the importance of studying particular convulsive seizures, due to spasm of certain groups of muscles which have especially important duties, we must, as regards treatment, consider the more general question of the state of the child's health or nervous system, which permits occasional spasm of muscles anywhere, whether these be of the limbs, of the thorax, or of the larynx. The inference is that the causes which give rise to laryngismus are essentially similar to those which give rise to other varieties of Convulsion. (See p. 263.) It is convenient, however, to say a few words here on causation.

The general belief is that this form of Convulsion is oftener than other varieties of Convulsion determined by the irritation of dentition, a belief which the facts as to age seem to justify. I say "seem," because the eruptions of the teeth are no doubt to be considered as marks of stages of development of the whole system, just as the occurrence of menstruation is later in life. Further, there is another fact of very great importance never to be lost sight of, viz. that, as Jenner, Elsässer, and Gee have pointed out, children the subjects of laryngismus are usually, almost always, rickety. Forty-eight out of Gee's fifty cases were unquestionably rickety, and in the two exceptional cases there was laryngeal catarrh.

We must at least modify the inference as to the influence of dentition, and say that the irritation of dentition produces laryngismus in rickety children. In none of Gee's cases (op. cit.) was there any reason to believe that the teeth bore any part in the causation of the fits. In accordance with these facts and opinions, whilst we must certainly endeavour to remove every source of eccentric irritation, we must also treat the child for rickets as well. "Laryngismus, when treated as if wholly dependent on the rickets, even if it be not so in fact, ceases to be a serious disease" (Gee).

Laryngismus has been attributed to enlargement of the thymus, but this view is not now entertained, one very good reason being that post-mortem examinations show that in many instances there is no enlargement of this organ. Moreover, in cases where a large thymus has been found there has been no laryngismus.

Unilateral Convulsions and Hemiplegia.—The Convulsions are limited to one side, or they affect one side first and chiefly. In such a fit, if it be severe, the face, arm, and leg of one side are in spasm; the head and both eyes turn to the same side, and next the chest becomes fixed. When severer still, the other side of the body becomes affected in the same way as the first, but to a less extent; the spasm may return to the side first affected. In this class of fits the spasm may be, for a while, very limited, *e.g.* a few jerks of the head to one side, or to spasm of one side, or it may be sometimes limited, and may at other times affect the whole of one side, or spread over the whole body. When the spasm is very limited, to the arm for instance, or even, when slight in degree, to one side of the body, there may be no loss of consciousness. The sources of danger to life from the paroxysm in this and in other forms of Convulsion are either that the spasm may fix the respiratory muscles, or that the frequency of the attacks may severely exhaust the child.

It is important to note this kind of Convulsion, because it is the one which is often followed by hemiplegia. We shall therefore anticipate what has to be said of the sequelæ of fits so far as this symptom is concerned. We should always carefully examine the child's limbs, after as well as during a fit; and when the fits of this kind are frequent, we may find the arm and leg of one side paralysed. If however the child be deeply insensible, we may not be able to determine this, just as we cannot determine the existence of hemiplegia in some cases of cerebral hæmorrhage in adults so long as the patient is very deeply comatose. We may first find out that the patient is paralysed when he is recovering from the "status epilepticus." I believe hemiplegia is common, but the palsy usually passes off quickly. This is precisely what occurs so often in adults. We frequently see unilateral convulsions, or more strictly convulsions beginning unilaterally, in adults, followed by transient hemiplegia—the epileptic hemiplegia of Dr. Todd. However, the palsy in children sometimes does not pass off; and if it remains for many days *after convulsions have ceased*, and if, above all, it remains so long after but *one* severe convulsion in a child otherwise seemingly healthy, it is very likely to be permanent. The palsy may be only a little weakness, or there may be complete immobility. In most cases the leg at all events recovers so far that the child can walk. As the child grows up, the condition is often a mixture of palsy and spasm. There is either "contracture" or "spastic rigidity" of the hand and foot—the foot suffering very much less than the arm. More rarely the face suffers too in the same way. As the child grows up the paralysed parts are smaller than those of the other side, the bones as well as the soft parts; the scapula is frequently strikingly smaller on the paralysed side. The condition is not like that of limbs affected by infantile palsy. The muscles respond to the interrupted current, and when there is much spastic rigidity, the arm, although shorter than the sound limb, may be thicker. Here we may say a few words as to the cause of this symptom.

In the first place, unless the child's nervous system is altogether different from that of the adult, the symptom points to disease of the opposite side of the brain. (*Vide infrà* on Convulsions in Adults.) But such a symptom does not of course point to any particular pathological change.[1] It is rare to find atrophy of the optic nerves in hemiplegic children, and this is some evidence that there is no gross lesion, such as tumour, tubercle, &c. The changes are probably minute. The causation of the symptom cannot be clearly discussed. But this issue may be raised: Is it the result of the very same changes which caused the Convulsion, or is it the result of damage to the brain in the paroxysm, *e. g.* to excessive congestion, or even rupture of vessels from sudden stoppage of respiration? I have no doubt it is owing to the first cause. 1. Because the rule is that the spasm has been on the side afterwards paralysed, or has begun on that side and affected it chiefly. 2. Because in the epileptic hemiplegia of adults, in whom we *do* sometimes find gross changes, syphiloma for instance, we find the disease in the cerebral hemisphere opposite the side of the body, first convulsed and afterwards paralysed. To this may be added the argument that general congestion of the brain is not likely to lead to so local a symptom—to paralysis of *one* side of the body. The fact that the hemiplegia is often transient does not show that there have been no local changes, because in adults, hemiplegia, after a convulsion, is often transient, even when there is organic disease of the brain,—syphiloma for instance.

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[1] I have made an autopsy on the body of a young woman, twenty-two years of age, who had been hemiplegic in the left side, after one series of fits, since the age of about three years, and subject to frequent convulsions from the age of five or six. The right cerebral hemisphere was much smaller than the left, and the left arm and leg were smaller than the right arm and leg. I found, however, no disease beyond what the unilateral atrophy implies. I have to thank my friend Mr. Norton for permission to see this patient.

The Convulsions are general.—It is not meant that the Convulsion affects both sides together, nor both sides quite equally, but that both sides are nearly equally affected, and nearly at the same time. The chief point here is to consider the condition of the thorax in the paroxysm. A long stoppage of respiration is the worst symptom we can witness, and when a child dies in a fit he is no doubt killed by the prolonged fixation of his thorax. In some of these cases the limbs seem to be comparatively little affected, although all four are somewhat affected. The older the child, the more the limbs and the less the chest suffer. These fits vary very much in degree of severity. If slight, the child may, as in other varieties of Convulsions, seem quite well shortly after. If severe, he may remain exhausted and appear dull only; if very severe, he lies in deep coma. The frequency of the fits modifies his condition. He may have a second fit before he has recovered from the effects of the first, or he may be, as the nurse will say, "in and out of fits all day long."

The child may have an attack and never suffer again. He may die in his first fit. It is not very uncommon for a fit to occur in a child who is seemingly quite well. He is suddenly convulsed, and may as soon as the fit is over go to play again as if nothing unusual had happened. It is to be insisted on that, however well a child may seem before and after a convulsion, we cannot be sure that he will not have more. He probably will. Next day or next week he has another, and then perhaps thirty in the day. From a rapid succession he suffers in two ways:—1. The respiratory function is much interfered with, and it is suddenly interfered with. 2. There is very great exhaustion from the severe muscular "exercise," and want of sleep. These things will be particularly referred to under "Treatment."

SEQUELÆ.—The chief sequelæ may with great looseness be arranged as, (1) Paralysis; (2) Amaurosis; (3) Defects of Speech and Disorders of Mind; (4) Squinting; (5) Paralysis of cranial nerves. There are of course other defects after Convulsion: loss of smell, loss of hearing, and unsteadiness of gait. These, however, are less common; they have different significance, being more accidental than the other defects I have named, and often depend on organic disease.

(1) Now children are subject to two kinds of paralysis, one of which almost deserves the name of essential, and is well enough recognised when called Infantile Palsy. The other, which will occur at any age, namely hemiplegia, is the form of paralysis which most frequently follows Convulsion, and has been already considered. (2) Amaurosis will be considered elsewhere: it is a rare sequel of Convulsion in children. If we find double optic neuritis, or double optic atrophy, we fear there is a gross intracranial lesion, such as tumour, a lump of tubercle, &c. Recovery from a condition very like that occurring from meningitis does not contra-indicate the existence of gross organic disease, if there has been double optic neuritis. Atrophy of the optic nerves occurs with fits owing to chronic hydrocephalus. Under any circumstances we can do nothing for Amaurosis from atrophy of the optic nerves. (3) With loss of speech we may take in mental defects, because in children the two things often go together. (Deaf-mutism is not considered here. If deafness occurs from any cause before speech has been acquired, mutism is the result.) After attacks of Convulsion, children are liable to lose their speech, and this loss occurs without any notable lack of power in the articulatory muscles. The proof of this is that the child eats and swallows well, &c. After a while, a few months or a few years, he begins to utter some word or syllable, *e.g.* "do, do," "ta, ta," "mam, mam," and he utters such syllables very distinctly. This loss may be temporary or it may be permanent. There is rarely hemiplegia along with loss of speech in children, or at all events rarely persistent hemiplegia. Sometimes after Convulsion, before speech is acquired, the child never learns to talk. If the child is only beginning to talk when the fits occur, loss of so recent an acquirement is usually only temporary.

In most of the cases I have seen where the loss of speech is permanent, the children are spiteful, vicious, and they have nearly always uncontrollable tempers. Children may be left after Convulsion in this general condition without loss of speech. These are, I think, the saddest cases in the whole range of our practice. Trousseau says that idiocy very often supervenes on infantile Convulsion. But the condition of speechless children is often worse than mere imbecility. There are many varieties of this sad mental condition, from some vulgar coarseness of mind to a total absence of decency. No purely medical treatment is of any avail. In many cases a false sentiment induces the parents to let the enfeebled mind grow as it lists rather than put their child to the annoyance of vigilant discipline. Indeed sometimes a fond mother not unnaturally mistakes a kind of animal vivacity for cleverness, and will not be persuaded that her child is much different from others, even when he cannot or will not dress himself or cut his food. Strenuous efforts must be made to teach the child to talk, for Dr. Langdon Down tells me that idiots who have come under his care speechless have been taught to speak. Very strangely, the worst of such patients even when speechless have often a good ear for music, and will hum or sing tunes correctly. This is not an important sign of mental power. Dr. Down tells me that idiots often can sing. The younger the child, the more likely is loss of speech to be attended by general mental deterioration. I do not know how it is that there is loss of speech after some convulsions and not after others. It is not, I believe, the result of the Convulsion, but is another result of the disease in the brain, be it what it may, which gives rise to, or permits, the Convulsion. Loss of speech is indeed a rare sequel of Convulsions, and will occasionally attend a simple attack of hemiplegia—*i.e.* simple in the sense that it is unaccompanied by Convulsion, or other evident symptoms. I may just observe that the association of defects of speech with symptoms pointing to disease of the left hemisphere is not so striking as it is in adults, but it will be found that they more often occur with Convulsion and paralysis of the right than of the left side of the body.

(4) Squinting has long been considered a common symptom of affection of the nervous system in childhood. But Helmholtz has shown that the most frequent cause of strabismus is a congenital defect in the eyeballs. It is found in those whose eyes are hypermetropic. Of course a child may have strabismus, as an adult may, from paralysis of one or more of the muscles moving the globe. It is generally held, however, that strabismus, when a nervous symptom, is a result of spasm of muscles. Yet neither paralysis nor spasm of one muscle—for instance, of one internal or of one external rectus—will properly account for the common form of strabismus. The kind of strabismus we meet with in children during or after Convulsions is not found during nor after Convulsions in adults. The presumption is that this difference is owing to differences in the development of the nervous system of the child and the adult.

(5) If the whole of the muscles supplied by any one of the cranial nerves are paralysed during or after Convulsions in a child or in an adult, we are certain that there is disease of that nerve trunk, or of the part of the nervous system where the nerve issues. In these cases we are usually able to infer that there is organic disease. We should especially inquire for evidence of syphilis.

THE CAUSES OF CONVULSIONS.

We have considered the several kinds of paroxysm, and now we have to speak of the diagnosis in cases in which a paroxysm of any kind occurs, or, as it is usually said, on the "causes" of Convulsions. However, we have already seen that we know scarcely anything of the pathology of children's Convulsions; we do not know what is the change in the nervous system which produces this symptom, because in most cases the brain appears healthy *post mortem*. It is scarcely fair, therefore, to use the word "cause." We can, however, group cases according to certain circumstances, and the following list will show, better than any exposition can, the limited way in which the word "cause" is to be used with reference to Convulsion. It is needless to say that the arrangement is a very arbitrary one, but it is convenient as guiding us in prognosis and treatment. We shall find that many a case will refuse to be placed altogether under one of the headings. For this reason it is impossible to discuss the points raised under each of the five following headings, quite separately from those raised under the others. When called to a case of Convulsion, we may find—

1. That an acute illness is setting in, as, for instance, an exanthem or acute cerebral disorder.

2. That the Convulsion is one of a series of symptoms, the result of organic disease of the brain or of its membranes, such as tumour, abscess, syphilis, &c.

3. That the fit is part of some chronic condition of ill-health, such as rickets, exhaustion and emaciation with diarrhœa.

4. That the child is suffering from some slight and temporary thing, such as an overloaded stomach, perhaps diarrhœa, the irritation of worms, or the irritation of teeth.

5. That the fits are of a kind which, for want of better knowledge, we are obliged to call essential, eclamptic, or epileptic.

(1.) There are symptoms of fever. Here we should fear that some acute illness, as pneumonia, small-pox, or scarlet fever, is beginning, of which the Convulsion is, if not the first, yet the first prominent symptom. It scarcely comes in my way to do more in this paper than urge the recognition of these rarer possibilities in the crowd of more probable causes. I must refer to special articles on the acute diseases of children. We should always make investigations for many acute diseases when there is heat of skin. The chest should be examined as a matter of routine, as not very rarely a child is first found by its parents to be ill when a convulsion occurs, although when we examine we discover that there is bronchitis, or even that one pleural cavity is full of fluid. The history of scarlet fever in other members of the family would be a most important guide, and we must never fail to make inquiries for it, however suddenly the fit may have come on in the most robust child. Dr. West mentions a case, no doubt scarlatinal, in which Convulsions succeeded by coma destroyed in a single day a healthy boy two years of age. We should in all doubtful cases examine the urine. The presence of blood in the urine would help us to determine whether the fits were or were not the result of masked scarlet fever. We require more observations on the condition of the urine in convulsive seizures soon after the attacks. Sir James Simpson has found albumen in the urine of a child three days old who suffered from Convulsion.

Feverish symptoms may be thought to point to causes which primarily affect the head itself, tubercular meningitis for instance. The difficulty in diagnosis is only likely to occur in cases where Convulsion is the first symptom, or, in more strictness, the symptom for which we are consulted. It is to be remarked, however, that Convulsion is rarely one of the symptoms of the invasion of tubercular meningitis, and when it is we usually find that the child has for weeks, without obvious cause, been getting thin, listless, and peevish—that in reality he has been ill for some time. Moreover, facts as to age will often help us in the diagnosis of many of these cases. Tubercular meningitis rarely attacks children under two years, and very rarely indeed under one year of age. It is well to remark here that we must not mistake the status epilepticus—the condition resulting from a quick succession of fits—for meningitis. If the convulsions be very numerous, the illness beginning by a convulsion, the disease is very unlikely to be meningitis.

(2.) Then it is possible that the Convulsion may be a symptom of organic disease, *e.g.* tumour. Such cases in children are very rare. There is usually a history of severe headache and urgent vomiting for weeks or months before the Convulsions, without any previous rapid emaciation to lead us to suppose there is tubercular meningitis. Moreover the symptoms are capricious, the headache and vomiting often ceasing for days. When these symptoms are not urgent we may be first consulted for a convulsion; we see cases occasionally in which a convulsion occurs from tumour of the brain in a child whose symptoms had been primarily so slight as not to prevent his going about, the severe headaches being put down by the child's friends to "biliousness," &c. In these cases we can only decide by considering the general evidence. The urgent, purposeless, often bilious, vomiting, possibly existing with clean tongue, and not infrequently with good appetite at intervals, days of comparative freedom from marked symptoms, point to the diagnosis of tumour. But the most conclusive evidence is the presence of double optic neuritis. If with headache, vomiting, and convulsion there is double optic neuritis, we may be almost certain that there is cerebral tumour. Double optic neuritis is rare under seven years of age, and very rare under five.

In all cases in which there is or has been discharge, especially offensive discharge, from the ear, especially when there is also pain in many branches of the fifth nerve; above all, if palsy of the portio dura nerve comes on, we should fear one of two things: (1) cerebral or cerebellar abscess, or (2) meningitis. It is not part of my task to enter fully into the diagnosis of these two conditions. We must not decide unless there are *general* symptoms also, *e.g.* increase of temperature, constipation, vomiting, and stupor, for discharge from the ear is not infrequently attended by *chronic* convulsions occurring at intervals for years (epilepsy), and the fit we are consulted for may be the first of such a series. What the connexion of the ear disease with the fits in these chronic cases is we do not know, unless we adopt the explanation that they depend on irritation starting from the ear. The frequent repetition of fits, even twenty or thirty in twenty-four hours let us say, does not make us incline to the diagnosis of abscess or meningitis, because it is not at all uncommon for Convulsions both in children and adults to occur in batches when there is no ear disease and no evidence of organic disease in the brain. The stupor which is produced by a succession of fits is of little moment, comparatively speaking, unless there has been before the advent of the fits severe pain in the head and vomiting, and unless the respiration and pulse be irregular.

It is possible that the Convulsions may be owing to congenital syphilis; but we cannot so conclude unless, *besides* a clear history of syphilitic taint, there are such symptoms as palsies of cranial nerves, or paraplegia. The existence of hemiplegia with a mere *history* of syphilitic taint would not warrant the diagnosis. Of course if signs of active syphilis be present, especially nodes, and especially if the patient be above three or four years of age, we should treat for syphilis. Indeed, in chronic cases at all events, if the patient's brothers or sisters show signs of inherited taint, we should give iodide of potassium.

(3.) If we found the child emaciated (there being now, we are supposing, no signs of acute mischief and no signs of organic disease), or if he had diarrhœa or dyspepsia, we should hope that the fit was but a sign that the nervous system was temporarily suffering with the rest of the body, and that it would cease to suffer when the child was brought back to good general health. Convulsions not unfrequently occur in infants improperly fed. In these cases there is often dyspepsia and diarrhœa, there is a big belly with general emaciation. The convulsions are then often ascribed to irritation from cutting the teeth, or, especially when there is diarrhœa, to the irritation of the intestinal canal. (The diarrhœa itself is sometimes ascribed to eruptions of the teeth.) These causes may have something to do in *provoking* the fit, but they cannot, I think, be solely to blame for it. Yet there can be no doubt as to the propriety of helping a tooth through if the gum be tense, swollen, or red; but to lance the gums as a matter of routine is not good practice. I have no belief that such irritations are the *sole* causes of fits; but I will now only deprecate exclusive attention to these supposed sources of eccentric irritation. If fits do sometimes cease when a vermifuge brings away worms, or when the gums are lanced, we know that in many cases they often continue when the system is freed from such cares, or at least when we have done all we can to get rid of them. I think with young practitioners, such "causes" are held to explain the occurrence of the fit so thoroughly that they keep in the background the evidence the child's general state will afford. We are, to say the least, unauthorized to ascribe children's illnesses solely to the irritation of teeth and the like, unless we have most thoroughly considered all other possible causes. Above all, we must negative the existence of rickets. In Gee's article, several times referred to, the frequent, almost constant, association of Convulsion, including laryngismus, with rickets, is insisted on. He notes seventy-three cases of Convulsion, and of these fifty-six were dependent on the "general condition" of the child. These fifty-six cases were, he tells us, of that kind commonly called, *par excellence,* cases of essential Convulsions (eclampsy). " Now, of sixty-one eclamptic children, fifty-six were rickety. Saying this I fear that I shall incur the charge of exaggeration. It is necessary to explain that my experience is wholly derived from the children of the poor." He tries to show, and I think he shows conclusively, "that the existence of a constitution leading to rickets is the most important fact in the kind of Convulsion [that depending on the general condition of the child] in question." And even when no other causes are obvious, we must not infer that the relation between teething and Convulsions is one solely of irritation transmitted from the gum to some part of the child's nervous system. The coming through of a tooth must be thought of as an outward mark of a certain constitutional progress in development, as the occurrence of menstruation is later in life. It is far better to acknowledge that very often we cannot find out what causes a fit than to put it down to an orthodox cause for the want of a more real one.[1]

When diarrhœa is severe the child is often emaciated, and the fit is more likely to be due to exhaustion than to eccentric irritation starting from the intestinal canal. We should not adopt routine efforts at clearing out the bowels to get rid of "undigested irritating" matters. In most of these cases the child has been improperly fed, and careful dieting is of very much more moment than immediate treatment of the diarrhœa by drugs. A child is at once overworked and underfed when it has to take into its stomach large masses of food which it cannot properly digest. We must certainly not consider the most violent convulsion in a child who is thin or who has exhausting diarrhœa as a result of " congested brain." There may be stagnation of blood in the head, the result of the paroxysm, but not any " active " process requiring antiphlogistic treatment. We do not know what the intracranial changes are which cause fits, and we have no evidence that cerebral congestion occurs *before* the attacks. We have plenty of proof, from disease and from experiments on animals, that Convulsions will follow anæmia; but as in these instances the anæmia is *sudden,* perhaps the facts are not quite to the point. We have, however, clinical evidence that they occur in feeble children and in the course of exhausting diseases. Since the days when antiphlogistic measures were so freely resorted to in children's disorders, " we have learned," Vogel says (op. cit. p. 385), " that pale anæmic children are as liable to be attacked by Convulsions as robust and plethoric ones." Beyond question the *violence* of fits and their *frequent repetition* are not the clinical signs of active changes in the head. Heat of skin and vomiting, headache, and irregular pulse are the real signs of acute changes in the head—encephalitis and meningitis. But since the brain may become much congested as a consequence of the fits when they are severe and frequently repeated, it is intelligible that some advantage may follow the application of leeches by a reduction of the congestion, although there is no inflammatory process to relieve. I say again that severe and repeated Convulsions are not the signs which should make us deplete generally, apply blisters, or give purgatives largely. Treatment of this sort is admittedly a most fatal mistake in those cases where the fit is but one sign of starvation, as it undoubtedly is in many cases of diarrhœa, and in cases of wrong feeding.

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[1] "Much *éclat* has lately been made in England and France with the scarification of the gums. Some recommend a crucial incision; others, the removal of the whole cap which covers the head of the tooth. But, as an admonition, it is premised in all the reports and laudation, that the tooth has to be very near eruption, otherwise the scarification will be of no benefit. I have frequently performed this operation, but have always found that the lancinated wounds of an inflamed mucous membrane heal very badly, and ulcerate for a long time; that the nervous symptoms continue notwithstanding, till ultimately artificial or spontaneous diarrhœa supervenes. Indeed, if we have to wait until the tooth is "very near" breaking through, then the process is in fact near its end, and any other simple remedy is as efficacious as this, which is attended by a considerable amount of pain." (Vogel, Raphael's Trans. p. 107.)

(4.) It does occasionally happen that a child is attacked by Convulsions when in apparently good health. But before concluding that a child is in good health we must, I repeat, consider very carefully whether or not there are signs of rickets, and we "must bear in mind that active rickets and the preservation of a large amount of fat are by no means incompatible." (Gee, op. cit.)

These are the cases in which it is plausible that the convulsion is the result of some temporary cause, such as over-eating; of some removable cause, such as the irritation of teeth during dentition; or of some sudden excessive mental disturbance, such as fright. I consider it very doubtful whether any of these so-called "causes" induce fits in children whose nervous systems are healthy beforehand. But it is very likely that they are exciting causes when it is not healthy. I have twice made autopsies on children who have died in fits after a meal, and in each case the stomach was full of food. In one case the child had had fits before, but none for three months before the fatal seizure; in the other the fit which the child died in was the first. If then we find that a child has had a fit soon after a meal which we have good reason to believe was large—say a basinful of sop—we should give an emetic. If we find that a tooth is coming through, we may justifiably lance the gum. But when we have done this we must bear in mind that a nervous system which has given way from such temporary and comparatively slight exciting causes, will be very likely to fail again when again tried by indiscretion in feeding or by eruption of teeth later in the dentition period. Indeed, we must, I think, conclude that the nervous system of a child cannot be healthy if a slight and temporary cause produces a convulsion, however healthy the child may look. We often find that the fits recur when we have done all we can to remove supposed sources of irritation. There are great differences of opinion with regard to the influence of dental irritation. Vogel says (op. cit. p. 387): "Eclampsia, originating from dental irritation, belongs to the serious forms, and often leaves behind it partial paralysis and imbecility." Meigs and Pepper, in their most valuable work on Diseases of Children, say: "As a general rule, the convulsions which depend solely on the process of dentition are slight, and last but a short time. In all the instances that we have seen in which this was the only cause to be detected, the attack was of this nature."

(5.) We will now consider cases in which Convulsion attacks healthy children without obvious cause of any kind, and cases in which they continue when we have removed all temporary sources of irritation. Once more having regard to the important researches of Elsässer, Jenner, and Gee, I would urge the consideration whether in these cases the "healthy" child is not rickety, although often slightly so. It is to be remarked that a child may suffer fits from blows on the head, and occasionally we see patients who have Convulsions after severe blows, followed by indentation of the skull. These cases it is not my task to consider. We are sometimes told by the friends of our little patients that palsy or convulsion followed an injury, but on inquiry we find there is not a shadow of evidence of a blow or a fall having occurred. The friends suppose very naturally that a fit must have a cause, and if their child has a convulsion in the midst of seeming good health, they infer that the child has had a fall. When we find a child the subject of Convulsions for which we discover no cause, or infer none from the condition of the teeth, bowels, and general health, we think of the terms eclampsia and essential. I do not, however, use these terms, for I know no means of distinguishing betwixt an epileptic fit in a child and an eclamptic fit. The practical point is this, and it is occasionally put to us by the child's friends: Is it epilepsy? I take this to mean: Is the illness one which will quickly, in days or weeks, run a course to death or to permanent recovery, or will the child recover from the fit or series of fits, but be liable to occasional attacks of convulsions for years or for life? This seems to me to be the practical question. Now, when we see a child in his *first* fit, we certainly cannot tell, whatever the age may be. The paroxysm is the same in all cases of general Convulsion. There seems to be great unanimity among authors that the eclamptic fit is quite like that of epilepsy. Niemeyer speaks of eclampsia as acute epilepsy. Vogel says it is impossible to distinguish the paroxysm of eclampsia from that of epilepsy. Then epilepsy will occur at any age. Vogel (op. cit. p. 411) states that Beau found, out of two hundred and eleven epileptics, that the disease was congenital in seventeen cases, and that it occurred from birth to the age of six years in twenty-two cases. Nevertheless, Vogel says, "Young children in general rarely suffer from true epilepsy, as we might expect, if the more frequent eclampsia be regarded as a distinct disease." And he adds: "Eclampsia is easily distinguished from the disease under consideration (epilepsy), by the fact that it almost always occurs at the breaking out of an acute affection only; that the general condition of the patient, after the termination of the Convulsions, is not restored; and that it is often fatal, while epileptic attacks are almost always devoid of danger." I submit that at the best we can only deal in probabilities. The older the child the more seemingly causeless the fits, the slower the succession—say a fit every other day, or fits scattered at irregular intervals of days or weeks—and perhaps we may say the less rickety the child the more likely are the fits to be epileptic, *i.e.* the more likely is the child to continue for years or for life subject to fits. But I am convinced that we can give a prognosis in no case with anything like certainty. I do not exempt cases where a child has had one fit during the eruption of a tooth or during an exanthem. We can only say, even in these cases, that the child is very unlikely to suffer again. If the child be partially hemiplegic after a seizure, he is very likely to suffer from Convulsions later in life.

Epileptic fits in adults not rarely date from Convulsions in infancy. The connexion is shown now and then by straggling fits at intervals of months or years, or by uninterrupted continuity of attacks at fairly regular periods. I have no facts, and I know of none on record, to show how many children keep well after getting through an illness with severe Convulsions, but I am certain that attacks in infancy —from one which attracted little attention, "we thought," says the mother, "it was only the teeth," to a whole batch—are occasionally followed by epileptic fits near the age of seven, fourteen, or twenty.

In reference to this question, it is important to ask if the child's near relatives have had nervous symptoms. Every medical man can relate instances of fits, or of other symptoms of cerebral disease in different members of one family. I have had under my care a girl of eleven years of age, who has had fits from the age of six months. Her sister, three years of age, had had them from the age of one week; another sister, aged ten, from the age of four years; and a fourth, also a girl aged eight, from six years. Instances so striking are rare. The fact that several of the child's relatives have had hemiplegia or Convulsions from embolism, clot, syphilis, &c., has no bearing whatever on the question. If the child's brother, or sister, or mother had Convulsions in childhood, the evidence is perhaps stronger; but I confess that I have very little faith in the hereditariness of such *symptoms* as epilepsy or Convulsions. The occurrence of Convulsions in several children of one family may be because they all suffer from rickets. We may believe that a "nervous temperament" is transmitted, but if so, there will be a predisposition to many nervous affections, and not to one symptom only. At the best it is very difficult to obtain certainty as to hereditariness, as Convulsions are so very common in children, and occur, it is presumable, from numerous pathological processes.

PROGNOSIS.

The prognosis of a symptom with so uncertain a meaning must, of course, be very uncertain, and much has been incidentally said on prognosis. Here may be excluded from consideration the attacks which precede or occur in the course of acute disease. The writers of other articles will speak of seizures so occurring. When a child has had a fit, and appears to be in good health again, the question as to the cause of the fits, with a view to forecast the child's future, becomes again urgent. If there is clear evidence of some source of irritation, and we have got rid of it, we may hope the child will keep well; but I have already spoken of the uncertainty of our hopes in this respect.

By far the most important question in prognosis is whether or not the child is likely to get through an attack or a series of attacks of acute Convulsions with life. In the first place, a single attack may be fatal, but this is a rare event. Wilks relates two striking instances of rapid death with Convulsions. One was a small, delicate child, six weeks old, who was seized suddenly with diarrhœa and Convulsions, and died in a few hours. The other patient was a child four years old, strong and healthy, who died soon after being brought to the hospital. In neither case was anything wrong found with the brain; but in the child four years old the stomach was distended with food. I have mentioned two cases of a like kind. Such cases, however, are rare. The consideration of less acute seizures is more important. We have no generalizations, and can only deal in generalities. The younger the child, the more likely is the result to be fatal. The more frequent or violent the seizures, the more profound the coma; and the worse the state of health in which the fits began, the more likely is the child to succumb. To consider the manner in which fits are likely to bring about death is the important matter. This will be considered with the treatment to which I now come.

TREATMENT.

It would not be correct, as I have remarked, to speak of any purely *rational* treatment of a single symptom, as it means things very different. Our treatment is nearly altogether empirical. And, of course, we exclude entirely from present consideration cases in which Convulsion occurs in such diseases as scarlet fever, meningitis, &c. Even an empirical treatment of Convulsion would not be justifiable in these instances. Our thoughts or treatment go hand-in-hand with our investigations into cause. Principles of treatment apply to Convulsions of all degrees, from rolling of the eyes to complete seizures.

If we find the child in a fit, we can do little during the paroxysm. We should see that every part of its dress is loosened, that it has a plentiful supply of pure air, and we should direct that it be laid down and kept quiet. Vogel says that by sprinkling the face and exposed chest with cold water, we may succeed in inducing deep spasmodic inspiration, by which the danger of suffocation at least is lessened.

Then as to general treatment, in the intervals of the paroxysms. I begin with what may be called attention to immediate circumstances. We undress the child, and it is possible we may find that a pin or a needle is sticking in some part of the body, even in the child's head, penetrating the brain. A needle has been found in the liver of a child who died of Convulsion (see Trousseau's Clinical Medicine, vol. i. p. 343, Bazire's translation). Trousseau believes that blisters and mustard plasters are often the causes of fatal Convulsions.

If we find that the fit came on after eating, we should give an emetic of ipecacuanha. If a gum be swollen and tense, we may properly use the lancet. We should inquire after the state of the child's bowels; if they are constipated, we should give a purgative. But none of these things must be done as matter of routine. The presence of diarrhœa, especially, with tenesmus and expulsion of little but mucus, may show that there is irritating matter in the intestinal canal, and it is then proper to give a dose of castor oil. This must be, however, only to make a starting-point for careful dieting. The presence of abdominal pain and constipation in robust children may lead to the suspicion of retained fæces. Diarrhœa, which is—paradoxical as it may seem at first glance—sometimes a sign of constipation, would not prevent the moderate administration of purgatives or enemata. For a child at the breast, an enema of an ounce of warm water or of thin gruel may be administered; at one year, two ounces. Very likely the diarrhœa is due to wrong feeding, and to diet the child would then be the most important thing to attend to. When the motions are very frequent, and if the child be thin and weak, we may try to check the diarrhœa by astringents, if proper dieting does not arrest it.

Warm baths are frequently used, and, when there is no great heat of skin, and no thoracic complication, the child may be put in a bath at the temperature of about 96° Fahr. for from five to ten minutes. Under any circumstances the feet and legs may be immersed in warm water. Then mustard plasters—a mixture of mustard and flour—may be applied to the calves of the legs for five, ten, or fifteen minutes.

When we have done all that immediate investigation prompts; when we have attended to the bowels, lanced the gums, ordered proper food, the Convulsions may persist, and may even increase in number and in severity. We are thus urged to do something more.

There is in attacks of Convulsion a tendency to (1) death by exhaustion, from the frequency of the fits and want of sleep; (2) death from asphyxia, from sudden and prolonged fixing of the chest walls, and from slow congestion of the lungs. The latter is often rather a way of dying than a cause of death. Adults die from convulsive seizures in each of these two ways.

1. If the child were much exhausted by frequent fits, or if he were weak to begin with; if he were thin, if he had long had diarrhœa, we should look most carefully to his support: we should prescribe beef-tea or juice of meat in abundance. Nor should we hesitate to give stimulants. The circumstances that would guide me most on this point would be the great frequency of short Convulsions, or constant startings, or frequency of other abnormal muscular actions.

The main object in treatment of disordered function of the brain in general, *e.g.* sleeplessness, delirium, and frequent, slight Convulsions, is to produce sleep, and to accomplish this we should give nutriment liberally; and if this fails, stimulants freely. I have already spoken of the importance of recognising that violent or frequent Convulsions do not depend on inflammatory changes. If the beef-tea or juice of meat were vomited, I should give milk with a little ice, and inject the tea and the juice.

Affusion with cold water has been advised, but this I should not adopt unless the child were robust. Of course I speak of cases in which there is no general fever, and no sign of inflammation of the membranes. A thin, delicate child I should keep warm. Vogel, however, says that affusions of the head with cold water, performed every hour or two, are useful against all Convulsions in children (op. cit. p. 107).

Then as to drugs. Antispasmodics have been given, but I think the best antispasmodics are nutrients and stimulants when these can be taken and digested. It is of course comparatively easy to get drugs into the stomach, but they may be vomited. It may then be desirable to give an enema of asafœtida, *e.g.* 20 to 30 minims of the tincture in an ounce of warm milk. In all cases—Convulsions in exanthematic and other acute diseases excepted—I should give bromide of potassium in large doses, by enemata, if necessary, if the fits were frequent, or if they continued several days. A mixture containing hyoscyamus, two or three drops for the age of six weeks, may be given with peppermint water. The great point, however, is, I repeat, to get the child to sleep, and to do this it is, I think, justifiable, simpler nutrients failing, to give stimulants freely. I should, however, be most wishful to give as much nutriment and as little stimulant as possible. Nor, of course, should I give wine because a child had a fit, but only when the child was feeble to begin with, or was exhausted by the frequency of the attacks, wearied too from imperfect sleep, and perhaps starving because the friends had not given enough nutriment before we were called. I should not give stimulants if the thermometer showed a great increase of temperature. Supposing nutrients and stimulants and the drugs mentioned were taken and retained, and failed to stop the Convulsions and to procure sleep, I should then venture to give narcotics. In no case should I prescribe narcotics except when I had ascertained that the child had previously taken nutrients and stimulants, or unless the child was fairly vigorous to start with. In no case, at any age, would I give opiates when there was great excitement without vigour. It is as hurtful to give opium at this extreme, as in the condition of brain which occurs with general febrile states, and which condition is supposed to be due to congestion. Nor would I give a narcotic more than once in twelve hours, and then I should give a decided dose, *e.g.* a quarter of a grain of Dover's powder under the age of three months; half a grain to a year, and a grain to a year and a half. Chloroform has been used by

Sir James Simpson, and, when the above ascending series of remedies—nutrients, stimulants, bromide of potassium, and opiates—have failed, this may be tried. Dr. West says: "In cases where depletion is inadmissible, where the Convulsions are not obviously due to organic disease of the brain, while they are both severe in their character and are returning with frequency, the inhalation of chloroform sometimes altogether arrests them." It is also, he says, of service in Convulsions of a more chronic kind. He tells us, however, that its effects are evanescent; he adds, that he has never seen mischief from its use. "It requires the constant presence in the house of some one competent to administer it."

2. Now I come to speak of the cases where the severity of the individual fits threatens death by asphyxia, or when from the breathing we fear the blood is largely delayed in the lungs. When Convulsions occur in robust children, bleeding is sometimes advised for them. It is a remedy which has been urged by many writers, but does not seem to me—I speak very respectfully—to be likely to be of use; but I have never tried it in any form, either by leeches or otherwise. For I have no faith whatever that Convulsions depend on any increase of nutritive changes that we can arrest by taking blood. There is in children's Convulsions no certain evidence as to the pathological condition of nervous organs. It is, however, often evident enough in robust adults, as when epileptiform seizures follow on blows, and on tearing up of the brain by mechanical injury, or the irritation of tumour, that bleeding could do no good—no good by acting on the cerebral circulation. But it would, I think, be good practice to relieve the venous system when, after repeated fits, the circulation is becoming embarrassed by great congestion of the lungs. Death seems, in these cases, to result from pulmonary congestion. Indeed, I think we neglect to bleed as often as we ought to do, on the principle Markham has laid down. I should be entirely guided by evidence from the colour of the lips and the state of respiration, and not by the heat of skin or by shivering. However, difficulty of the respiration so great as these signs imply would mark the advance of a fatal issue, and our treatment could only, I fear, be expected to delay it: when I did deplete, it would be by leeches only.

I have just spoken on the supposition that the fits are frequent and are running a rapid course, and are tending towards death by exhaustion or by asphyxia; but if the fits come on at intervals, as once every day, or once a week, and if from inability to discover what they really did mean, *i.e.* if unassociated with debility, irritation of teeth, &c. we were obliged to class them as epileptic or essential, I should adopt the same treatment as for epilepsy in the adult, supposing of course all general indications fulfilled. I should give bromide of potassium, a quarter of a grain under six weeks, half a grain under three months, a grain above, and a grain additional for every year. Indeed, the bromide is a most useful drug in chronic convulsions from any cause, and in most cases—excepting temporary and acute states like uræmia—I should prescribe it when other remedies failed, whatever was the state of the child. Since, however, there is manifestly an association of Convulsions with rickets in very many cases, it is well to treat the child for this condition in all chronic cases. I shall therefore conclude what I have to say on the treatment of Convulsion by the following quotation from a paper on rickets, by Gee (St. Bartholomew's Hospital Reports, vol. iv. p. 79):—"*Treatment.* This must be radical. And in cod-liver oil we possess a pharmaceutical agent worthy of a place beside iron, Peruvian bark, and mercury. We ought to lose no time over the symptoms of rickets; slight catarrh, diarrhœa, paleness, a tendency to fits, these will all disappear under cod-liver oil. Give expectorants, purgatives, styptics, and the rickets will increase under our eyes; nay, occasionally it will even develop *de novo* while a child is being treated for coughs, deranged bowels, and other apparently simple disorders."

Convulsions in Adults.

It is a matter of exceeding great difficulty to write on the subject of Convulsion in the adult. We may arrange Convulsions as they are local or general, as they are acute or chronic, as they depend on acute states, such as uræmia, on organic disease, such as tumour, or on changes which we infer to be functional. I shall follow the course adopted in treating of Convulsions in Children. I shall speak first of the varieties of the paroxysm, and then of the causes of the seizures.

There are no doubt innumerable varieties of paroxysm, but for clinical purposes we may arrange most of them in one of two classes.

Class I. The Convulsion begins unilaterally, and begins deliberately. In this kind of seizure consciousness is not always lost, and when it is, it is lost *after* the spasm has begun.

Class II. The fit begins either without any warning or by a very vague one, such as a strange feeling in the head ("giddiness," "swimming," &c.), or by a sensation at or near to the epigastrium, which is variously called "sinking," "faintness," "sickness," "fear," and sometimes by children "stomach-ache." In these cases the spasm is more contemporaneous, *i.e.* it affects the two sides of the body more nearly at the same time, and it affects them more equally. In this class of cases loss of consciousness is either the first symptom, which occurs very quickly after the first warning.

It is not pretended that there is an absolute distinction, for in the first class the spasm, in severe cases, spreads all over the body. In the second class it is not usually, probably never is, strictly equal on the two sides. Nor is it strictly contemporaneous; one side almost always, if not always, suffers not only *more* but *sooner* than the other. Moreover we often do not witness the fit, and we may be unable to learn how it began and how it affected the patient. Fits of the first class are almost always chronic. Those of the second also are mostly chronic (epileptic), but frequently acute; they may be symptoms of uræmia, cerebral hæmorrhage, &c. In handling the subject clinically we must arbitrarily put in the second class cases in which we can only learn that the patient has had a severe convulsion. In strictness, Class II. is merely a grouping of cases which agree in that Convulsion is the most prominent symptom. We must make a provisional arrangement for clinical purposes, and the above distinction is at least convenient in practice.

The paroxysms of the first class only need be described at length. These fits are far simpler than those of the second class. The patient can tell us more about them; we can, when present at a fit, obtain a clearer idea of what takes place, because the progress of the Convulsion is more deliberate than that of the second class. It is for this reason that I speak at most length on the "causes" of Convulsion, after describing the peculiarities of the paroxysm and its sequelæ in this class.

CLASS I. THE CONVULSION BEGINS UNILATERALLY.

Onset.—The patient or his friends tell us that he is subject to fits beginning by "working" in the hand, or in the face, or in the leg; sometimes there is a local sensation, often named an aura, before there is visible movement. The rule is, that in these cases the spasm starts in the very same place in each seizure. For instance, one patient's fits will always begin in his right index finger, another patient's always in his right great toe. The exceptions occur chiefly in cases of syphilitic disease of the brain. Taking a large number of cases, there is an order of frequency of onset to be stated. The spasm begins most frequently in the hand (usually in the index finger and the thumb), less often in the side of the face and tongue, and rarely in the foot.

Range (*Fits partial or general*).—In the severest fit the spasm first spreads over the side it begins in, then extends to the trunk, and then to the face, arm, and leg of the other side. It is important to observe that the spasm may stop at any stage. There are all degrees, from a slight twitching of one finger to general and severe convulsion. In other words, these fits may be partial or general. Moreover, the same patient may on one occasion have a fit limited to the hand or arm during which he is quite conscious, and on another occasion the spasm may spread all over his body; he will then become insensible, and may bite his tongue. The importance of studying the partial fits is that sometimes they occur for months before a severe fit. A few minutes' steady cramp—not "live blood," tremor, fidgets, or irregular jerks of a choreal kind—on one side of the face, in one hand, or in one foot, demand almost as serious consideration in prognosis as a severe fit does; for a patient who has such local spasm will probably suffer from severe convulsions.

Ligature.—These are the cases of "epilepsy" in which the fit may often be stopped by tying something round the limb above the part in which the spasm begins (or in which some abnormal sensation (aura) appears), by unclenching the closing hand, or by briskly rubbing it. As Brown-Séquard has insisted, such procedures are as successful in cases of organic disease of the brain, syphiloma for instance, as in cases where there is no evidence of such kind of disease. The patient or his friends may often arrest the progress of fits of this kind. We hear patients remark to this effect, "If I can get the hand open, I have no fit." I am convinced from what I have seen in Brown-Séquard's practice that a garter of blister above the part in which the fit begins will keep off fits of this kind. Dr. Buzzard has recently written an important paper on this method of treatment in the "Practitioner" for October 1868. In a few, but in very few cases, there is history of injury to the part in which the spasm begins.

Duration of the Attacks.—The duration of the attack varies much. It may last even ten minutes, but in such cases the spasm is long in spreading to the muscles of the thorax.

Consciousness.—These are the cases of Convulsion in which there is often no loss of consciousness whatever. The patient does not lose his consciousness when the fit is partial; the whole of one side may be involved in spasm without any insensibility, but the rule is that consciousness is lost at an earlier stage. For instance, when the fit begins in the hand, consciousness is usually lost when the spasm has largely involved the face, and when the head begins to turn. When it starts from the foot, consciousness is usually lost when the spasm or abnormal sensation reaches the body. There are in these seizures all degrees of impairment of consciousness, if such a phrase be permissible. The patient may assert that in some of his attacks he is conscious, but may qualify his statement by adding that he does not know people about him or where he is; at another time he may be profoundly comatose.

Affections of Speech (*Epileptic Aphasia*).[1]—In certain of these seizures the patient loses his speech without losing his consciousness. This is not to be confounded with *petit mal.* It most often occurs when the fit begins on the *right* side of the *face and tongue.* Occasionally there is only disorder of speech. Temporary loss or defect of speech is often observed even in cases where the spasm is limited to the right cheek and tongue. The patient may consult us because he occasionally suddenly loses his speech for a few minutes; for instance, he goes to a shop and on trying to give an order finds that he cannot talk, or he talks so badly that he is supposed to be drunk. We mostly hear that there is local spasm of the right cheek at the same time. (We rarely get any facts about writing in this condition; this part of the subject is too complex for consideration here.) It is hard to describe the *defect* of speech which sometimes *follows* these seizures. The defect is not, I think, quite like the defects of speech which are the result of small destroying lesions such as small clots, limited softening from embolism, &c. There is very much hesitation; the patient slurs his words and mumbles. I use the word defect, because there is never, so far as I have observed, permanent *loss* of speech (complete inability to utter words) after a convulsion beginning by deliberate spasm in the face or hand. If an adult does not talk at all for several days after a "fit," especially if he expresses himself quite well in writing, we should suspect malingering. If there be loss of *voice* with or without loss of *speech* after a "fit," pretence or hysteria is almost certain.

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[1] "There is a peculiar class of cases of epileptic hemiplegia, in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech." (Todd, "Nervous Diseases," Lect. xv.)

Epileptic Hemiplegia.—These are the cases of Convulsion in which there is so often hemiplegia—epileptic hemiplegia of Dr. Todd. Sometimes hemiplegia follows such seizures, and sometimes it does not. The presumption is that when the spasm is very severe—severe in degree that is, not necessarily, although usually extensive in range also—there is palsy. Thus much however is certain, that a patient who has fits of this kind (at all events when the spasm begins in the hand) can never be considered safe from hemiplegia. In very many cases the palsy is trifling, such a "numbness," for instance, as prevents the patient picking up a pin, although he may strike the table pretty strongly; occasionally there is perfect[1] paralysis. The palsy always affects the side in which the fit begins; and when there is *complete* hemiplegia, the hemiplegia is quite like that produced by plugging of the middle cerebral artery. When, however, the spasm is limited in range, the consequent palsy is limited in range. If the arm only be affected by severe spasm, the arm only is palsied when the fit is over; if the leg only, the patient drags it, but uses his arm well. Thus it happens that we may have complete palsy of the arm following a convulsion in which there had been no loss of consciousness whatever. Whether the palsy be complete in range or perfect in degree, it passes off in the vast majority of cases, and according to the degree of palsy in hours, days, or weeks—perhaps, however, leaving a little numbness—the patient may ever afterwards speak of his "weak side." Unfortunately, since the patient will doubtless continue subject to convulsive seizures of a like kind, we shall be obliged to admit that he will probably have the palsy again. Indeed he may have another fit even before the palsy from the former one has had time to pass off. More rarely hemiplegia is the first symptom, and Convulsion of the kind I describe occurs after or during recovery. The Convulsion affects first and chiefly the side paralysed. Since this order of events usually happens in cases where there is valvular disease of the heart, I suppose there is in these cases embolism of some part of the district of the middle cerebral artery.

Frequency.—In this class of cases the fits vary in frequency. They are, I think, more irregular in this respect than chronic convulsions of other kinds. The patient may have one a week, one a month, or he may have thirty or forty, nay literally hundreds, in a few days. When the fits are very frequent, the patient is usually, if not always, hemiplegic and deeply comatose. There is the "status epilepticus." It will be observed that in the paroxysms the spasm still affects first and more strongly the side already paralysed. The signs of danger in the status epilepticus are not so much the number of fits or the degree of palsy —the frequency of the fits of course adds much to the gravity of the case —as increase of temperature, abnormal respiration, and irregular pulse.

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[1] I use the words "perfect" and "imperfect" to express degrees of loss of power, and the words "complete" and "incomplete" to express differences in range of the paralysis.

CAUSATION.—A convulsion implies discharge of unstable grey matter. We have then three directions of investigation in these cases: (*a*) the seat of the changes in grey matter; (*b*) the pathological processes by which these changes are brought about; (*c*) the circumstances which favour the discharge (exciting causes).

(*a*) *Seat of Lesion.*—The probability is that in many cases we should discover no lesion *post mortem.* Although we must admit that grey matter is in an abnormal condition (because it discharges on slight provocation, and because it discharges abundantly), we must admit also that this abnormality does not involve any *great* alteration of structure. If it were much altered, even disorderly functions would not be possible. We are therefore not likely to *discover* the changes in the grey cells to which exaggeration of their normal function—to store up and expend force—is due. Niemeyer (op. cit.) says, "Experience teaches that the lesions from which abnormally active impulses proceed are insusceptible of anatomical demonstration."[1] Since increase of function, even in disease, implies increased nutrition, we infer that the grey cells affected in Convulsions store up force in large quantity, and reach a high degree of tension. Further, since they discharge on slight provocation—possibly even in periodical normal changes in the body, when by continuous nutrition a certain degree of tension is reached,—we must suppose they are in a state of highly unstable equilibrium. Instability, in this article, is made to include two things—nigh tension and very unstable equilibrium. But admitting that we cannot, or have not yet discovered the change which permits this duplex condition, we have ground for inference as to the position of the changes of instability.

In some cases we discover gross changes. (For convenience we shall suppose the gross change to be, as it often is, a syphilitic nodule.) The gross change affects the cerebral hemisphere[2] opposite the side of the body in which the spasm sets in. Moreover, in all the autopsies I have seen the disease has been in the region supplied by the Sylvian artery, and has affected convolutions—parts rich in grey matter. But the nodule we find is of course not the direct cause of the seizure—the seizure is the result of a discharge of grey matter, and the nodule is an overgrowth of connective tissue. In other words, the changes in grey matter on which the Convulsions depend are secondary to the nodule. Now, at least two views may be held as to the seat of these secondary changes. The view generally adopted is that they are in the medulla oblongata, and that they are the result of an "irritation" starting from the nodule, or rather from its neighbourhood. Another view is, that the grey matter changed is near the tumour itself, or in its vascular territory at least. Arguments in favour of the latter view are, chiefly, first, that the muscles first and most affected in the seizures are those most and longest affected in hemiplegia due to the disease of the corpus striatum; secondly, that the epileptic hemiplegia, when complete, is quite like that which results from plugging of the middle cerebral artery. Other reasons could be given. The occurrence of temporary defect of speech in certain of the seizures (*vide* p. 278) is of some value in localizing. It may, however, be said that disease of certain convolutions often causes no symptoms at all, and therefore that when Convulsions occur along *with*, they cannot *depend on*, changes in the convolutions. The word disease is, however, here used vaguely. It is true, as every surgeon knows, that much of the convolutions may be *destroyed* without the supervention of symptoms. The patient can *do without* certain parts of his brain, but if much of its grey matter be very *unstable*, he must have symptoms, for it will discharge strongly, and of necessity put muscles in disorderly movement. It may be asked, How is it, if the patient can do without the part which discharges, that there is sometimes hemiplegia? The explanation is admittedly speculative. The hemiplegia comes on *after* the paroxysm. Suppose the fit to begin by discharge of unstable grey matter in a certain convolution, the violent impulse thus originated will probably discharge lower and yet related centres of healthy grey matter. At all events, the nerve fibres to the muscles, and the muscles themselves, are suddenly in excessive function. I suppose, then, that the hemiplegia remains until the normal conditions of these suddenly overworked parts have been slowly restored by nutrition. Wilks (op. cit.) says the paralysis follows by "an inhibitory action."

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[1] "The proximate cause of convulsions is an abnormal increase in the nutritive changes of the nervous centres." (Russell Reynolds "On Epilepsy.")

[2] Wilks says ("Pathology of Nervous Diseases," Guy's Hospital Reports, 1866) "that the morbid conditions which we find to give rise to epileptiform convulsions are remarkably uniform. They all point to the presence of local irritation of the surface [of the brain]." Speaking of a case of epileptic convulsions in a patient who had tumour in the pons Varolii —a case which had been supposed to confirm Schröder van der Kolk's "supposition that the cause of epilepsy is seated in this part"—he says, "I have no hesitation in saying that for one such case fifty might be found in which the marked changes producing these symptoms occupy the surface [of the brain]."

As my plan, however, is chiefly clinical, I have discussed the question of position very briefly. It suffices for the discussion of the next question to admit, as we must, that there are changes of grey matter in *some* part of the nervous system.

(*b*) *The Pathological Process.*—The changes of instability may no doubt be brought about in many ways; but we shall limit ourselves to these questions: "Are the changes the secondary results of gross organic disease—tumour, for instance,—or are they minute changes, often inferable, rarely, if ever, discoverable?" If there are symptoms pointing to gross organic disease, we fear that the patient will die of that disease; if there are not, our fears are of a different kind, viz. that he will be subject to fits for years or for life, or, as it is often said, will be an epileptic. The first question then is, Is there gross organic disease?

If for no other reason than to economize space, it is well to consider first the symptoms which show that there is gross organic disease of *any* kind; and next, to consider the evidence by which we infer its particular nature,—whether, for instance, it be syphilitic or not. Before beginning our task, it may be well to point out that certain symptoms which young practitioners sometimes rely on are not to be relied on, either for or against the diagnosis of gross organic disease.

Tongue-biting is of no value in the diagnosis of the cause of any kind of convulsion: it is only a sign of severe convulsion. It occurs in cases of the first class described, if the fit be general and severe, and does not occur if the fit be partial and slight. It occurs in severe fits from uræmia, cerebral hæmorrhage, tumour, &c. Nor does the condition of the pupils during the fit furnish any evidence in diagnosis. Very great inequality of the pupils *after* the fit would point to organic disease. In these cases there may be, as has been remarked, no loss of consciousness when the fit is limited in range. It was said by the late Dr. Bright that absence of insensibility in convulsive seizures is some evidence that the lesion is organic. With very great deference, I must say I cannot accept this view. If we were to judge by post-mortem evidence alone, we might draw the conclusion; but we see patients who have convulsion without loss of consciousness, who not only show no signs of organic disease, but who, except for their seizures, seem to be in good health. Post-mortem examinations, in cases where there has been no evidence of organic disease, are very rarely had. I have not yet seen one. The fact that the fit is partial, let us say limited to the arm, the transitoriness of epileptic hemiplegia, the absence of fits for months, do not negative the existence of gross organic disease. It is to be especially insisted on that quick recovery from epileptic hemiplegia is of no value whatever in negativing organic disease, tumour for instance. The signs I have mentioned are of no value for or against the diagnosis of gross organic disease in this class of convulsions.

The evidence which warrants the diagnosis of gross organic disease in cases of Convulsion is of a different kind. We have carefully to distinguish betwixt the symptoms which are owing to local destruction of the nervous system and those owing to changes diffused in nervous masses about the destroying agent—the results of the irritation it excites. For instance, a certain form of hemiplegia is owing to the destruction of fibres and cells of the corpus striatum by, let us instance, a clot. This symptom is special to the part injured; injury to no other part produces it. Again, it is primary, for it comes on at once, from destruction of nerve fibres there seated. But, next, the patient suffers headache; his temperature rises, his pulse and his respiration become irregular. Such symptoms may be called general, because they do not point to disease in any one part of the encephalon, and they may be called secondary because they do not come on at once, but are indirect results of the irritation of the clot as a foreign body—of a local encephalitis. Hence we see that whilst the *nature* of the lesion matters little or nothing so far as the production of such special and primary symptom as hemiplegia is concerned—it suffices that nerve-fibres in the corpus striatum are by *any* means *destroyed*,

—it matters very much with regard to the general or secondary symptoms. The encephalitis in cases of clot is often a rapid process, but the same distinction is to be made in cases of tumours, syphiloma, abscess, and other kinds of gross organic disease. But it frequently happens that tumours, syphilitic disease, &c. occur in regions of the brain —in the cerebrum and cerebellum,—large parts of which may be destroyed without the production of any special symptoms—without hemiplegia or obvious mental defect. In other words, the patient does not suffer because the tumour has *destroyed* a certain part of his brain. He begins to suffer when a local encephalitis is excited by the destroying agent. And as this encephalitis does not always occur, he may have no symptom whatever from cerebral tumour.

What is the evidence which shows a patient the subject of a convulsion to have gross organic disease within the cranium? The symptoms which show there is organic disease within the cranium, not in these cases only, but in any cases—in cases of palsies of cranial nerves, hemiplegia, &c.—are such as severe headache, urgent vomiting, and *double* optic neuritis. The pain in the head has no value in diagnosis if it be the temporary sequel of a severe fit, or of a series of fits. We must be satisfied that the patient's "headache" is really pain, and not "confusion," "giddiness," "weight on the top of the head," &c. Nor can we lay stress on it as evidence unless it be intense, and unless it has lasted for some days or weeks. We can sometimes judge of its intensity by the patient's manner and by his expressions. He gives up work; he may remark, "it is not a common headache." He is said by his friends to "rave," and sometimes it is reported that he "knocks his head against the wall." The headache is of more value in diagnosis if it occurs in unusual places. If it be at the back of the head shooting forward, or on one side—I do not refer to nodes nor to neuralgic pain extending into the face—gross disease is likely. The vomiting is urgent, it is purposeless and capricious sometimes, for instance, occurring only at night or in the morning. The vomit is frothy "like phlegm," the patients sometimes say; and if there be very much retching, as there usually is, it is greenish or yellowish. The tongue may be quite clean, and the appetite may be good. Vomiting is not always present. When symptoms so well marked have lasted for several weeks, we suspect that the Convulsion is the result of gross disease. We may, I think, be quite certain if there is also paralysis of the whole of any one motor cranial nerve. If there be double optic neuritis as well, or its usual sequel, double optic atrophy, we may be almost absolutely certain.

Let us suppose we have satisfied ourselves that there is a gross lesion of some kind, we have now to find out what is its particular nature. We may, I believe, exclude clot in nervous centres as a cause of chronic seizures of this kind. No doubt effusion of blood on the surface of the brain would produce fits of this kind, and in all cases we should inquire carefully for history of injury to the head, and seek for evidence of chronic renal disease, the two chief conditions under which meningeal hæmorrhage occurs. I have only twice known albuminuria to occur with fits of this variety, and there is, I think, no warrant for the supposition that uræmia has anything to do with their causation. It is true that Convulsions called uræmic—after scarlet fever, for instance—are often unilateral, but as far as I know these seizures do not begin by a very deliberate spasm in one side. When they do, their real nature may be inferred by examining the patient's heart, urine, &c.

The gross organic disease may no doubt be of many kinds, but practically the point we wish to determine is, Is it syphilitic, or is it some other kind of new growth? It is needless to mention that when other symptoms of syphilis are present, such as nodes, the diagnosis of syphilis is almost certain, and needless to urge in all cases of this kind a very careful investigation for evidences of syphilis, such as scars on the skin, holes in the palate, white marks on the tongue. It is only necessary to speak of cases in which such decisive evidence is not to be had. In the first place the gross disease is frequently syphilitic. It is so with the very rarest exceptions when there is also double optic neuritis. It is next to certain that there is syphilitic disease if the patient has Convulsions of this kind along with complete palsy of the whole of any motor cranial nerve; for one great diagnostic mark of syphilis is that it produces random associations of symptoms. The evidence is clearer if the motor cranial nerve paralysed be on the same side as that on which the Convulsion begins, and on which there may be epileptic hemiplegia, because we are sure then that there are two lesions. (The facial paralysis, which is part of epileptic hemiplegia, is not of course included in the expression palsy of a motor cranial nerve, because it is not owing to disease of a nerve *trunk.*) If there is palsy of any nerve trunk, *e.g.* of the radial, palsy of one leg, or paraplegia, the great probability is that these symptoms and the Convulsions are owing to syphilis.

Still there may be some other kind of new growth, but this is very rare in cases of Convulsion of the class described, and rarer still when the Convulsion is attended by any of the other symptoms mentioned in the preceding paragraph. In some cases the recovery of the patient from local palsy, let us say of the third nerve, by iodide of potassium will make the diagnosis pretty certain. In other cases the length of time, *e.g.* many months or several years, the symptoms had lasted, would point to syphilis. There can be no doubt that we should treat for syphilis.

Fits of this kind occasionally follow blows on the head. In these cases we should carefully inquire for evidence of syphilis, as syphilitic disease of the brain is frequently "lighted up" by injuries. In some cases there is a depression of the skull on the side opposite the side of the body in which the fit begins.

Suppose now that there is no evidence of gross organic disease of any kind. In the vast majority of cases we can get no further, we can only infer that there are *not* gross changes in the brain, and as a corollary that the patient will not soon die, but will continue subject to fits. In a few cases there will be found evidence which will warrant the supposition that the plugging of small arteries of the Sylvian region is the cause of the pathological change.[1] It is certain that patients, the subjects of valvular disease, have seizures of this kind after or during recovery from hemiplegia. Yet although I have made post-mortem examinations of patients, the subjects of valvular disease, who have had Convulsions, I have had no post-mortem examination of one whom I knew to have had fits of the kind described. My supposition is that patients who have "epileptic fits" from intracranial aneurism suffer really from local embolism, and that when the aneurism is of the middle cerebral artery, or of some large branch of this vessel, the seizures will be of the first class. It is, however, held by some that the fit depends on irritation by the aneurism. Mr. Callender (St. Bartholomew's Hospital Reports, vol. iii. 1867) has made the very important observation that the "epileptic attacks belong to aneurism of the middle cerebral artery."

(*c*) *Exciting Causes of the Paroxysm.*—Some patients who are subject to fits of this kind are otherwise in very good health. Such cases are sometimes supposed to be owing to some very general cause. A patient who in the midst of good health has had a severe convulsion is naturally most anxious that his fit should be attributed to some very general and removable cause, and will dwell much on such facts as that he had taken something that had disagreed with him, or that he was in a close room, or will say that he was "bilious," or worried by anxiety the day it happened. Dyspepsia, over-work, fright, and the like, may be admitted to be factors in causation. I cannot however conceive that any such general conditions can alone produce fits which time after time begin in one hand and even in the very same finger even for months and years. In other words, I cannot conceive that they alone can determine the discharge of *healthy* nervous tissue in some particular locality. I class them as exciting causes, believing that there is some central change as well. Whatever view may be held, there is for therapeutical purposes a complete agreement that we should try to remove all such causes. We may find that the patient is dyspeptic. It is, I think, quite certain that in some cases the *paroxysm* frequently *comes on* when the patient is flatulent. So, although we may differ as to the way in which dyspepsia is connected with the seizure, there is no question that to treat the dyspepsia by careful dietary and by medicine is a matter of the very greatest importance. (See Dr. Paget, of Cambridge: Lectures on Gastric Epilepsy, *Lancet*, 1868.)

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[1] The inference is not that each fit or each series of fits depends on separate pluggings. It is true that sudden plugging of the middle cerebral artery, or perhaps of some large branch, may lead to a *severe* convulsion in a patient whose nervous system was previously healthy, but in these cases there is persistent hemiplegia after a fit. If a smaller branch be plugged and perhaps slowly occluded, the hemiplegia passes off or diminishes greatly, and, as before said (p. 278), occasionally the patients become subject to convulsion beginning in some part of the region previously paralysed. The hemiplegia depends on destruction of nerve fibres, the occasional spasm depends on instability of grey matter. It is evident enough that plugging will lead to destruction (softening even to diffluence) of fibres and cells, but it is not sufficiently borne in mind that plugging of small vessels may lead also to increased quantity of blood beyond the plug, and thus to altered nutrition and instability. At autopsies on patients who have died of or with plugging of the middle cerebral artery, whilst we find softening of part of the corpus striatum, we find also at the periphery "red softening."

These seizures, like other convulsive attacks, and other nervous symptoms, sometimes follow fright. The first fit of a series evidently depending on organic disease may follow fright so closely that we are driven to believe there is some relation betwixt the two things. I imagine that the fright merely *determines* the paroxysm which some other cause would afterwards determine. There is nervous tissue in a state of highly unstable equilibrium, which will surely discharge soon from some provocation, and now and then fright is that provocation. As no special point of treatment is involved, and as the discussion of such causes belongs rather to epilepsy, nothing further need be said here.

As to local irritation by worms, teeth, &c., all that need be said here is that we should try to remove these sources of irritation. Then possibly the part of the nervous system diseased, ceasing to be worried by such eccentric irritations, may cease to discharge.

CLASS II.[1]

Let us now suppose the patient's fit to be one of the second class. These cases cannot be considered on the same plan as those of Class I.; the paroxysms are too sudden, and the conditions under which they occur are too numerous and complex. In chronic cases of this class the same reasoning as to position, nature of change, and exciting cause will to a very great extent apply. We shall include in this class those cases of which we obtain no history of the mode of onset of the fit. For instance, we find the patient comatose, and we only learn that he has had a convulsion. It is then that the question arises, Is the fit epileptic? This is the great question when we see a patient in or soon after his first convulsion. But since we may find him hemiplegic, it will be best to use the expression epileptiform, and modify the question thus, Does the fit depend on a state of the brain or system which is such that the patient will recover from the fit in all probability to suffer similarly again and again for months or years, or is it owing to such causes as cerebral hæmorrhage, tumour, uræmia, &c., which will lead soon to a fatal result?

In the first place, the phenomena of the convulsion—it does not begin by deliberate aura in one limb—the nature of the paroxysm, stertor, coma, tongue-biting, furnish no reliable evidence. Cases of apoplexy from cerebral hæmorrhage are now and then diagnosed as cases of epilepsy, because the apoplectic condition was ushered in by a fit of an "epileptic character." It is to be insisted on that neither the kind of convulsion nor its repetition are signs serving in the diagnosis of the *nature* of the lesion. Neither enables us to say whether the fit is epileptic or not. Nevertheless it is freely admitted that in most cases we are right in the prediction we make. Although it is difficult to make a diagnosis, it is easy to guess. If we are called to a young man who has had a severe fit and who is not paralysed after it, and if we find that he has recovered or is recovering consciousness, we shall be right in the great majority of cases if we say, without any further medical examination, that the fit is one of epilepsy, and not one of uræmia, cerebral hæmorrhage, &c. But it is not necessary even to see the patients to make diagnoses which shall be *generally* right. And when we hear that a patient has been long subject to fits, from each of which he quickly recovered, or if we hear that he has had attacks of *petit mal* only before the convulsion, we shall be right in nearly all the cases when we make the diagnosis of epilepsy. But even under these circumstances the practitioner will be wrong now and then. For instance, there may be chronic renal disease, notwithstanding the patient has had fits of an "epileptic character" months before. Again, the former fits may have been due to aneurism of one of the larger cerebral vessels, and the fit we are called to may be owing to rupture of that aneurism. The former fits may have been owing to tumour, and the one we are called to may be the result of fatal hæmorrhage from that tumour. These are rare cases, but we are sure to meet with them now and then. If we do not consider these rare possibilities, we may make very painful blunders.

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[1] See page 276.

I repeat that a routine diagnosis of epilepsy in young people who have a convulsion will rarely be wrong, because such Convulsions are nearly always epileptic. And those who do not examine the urine unless the patient be dropsical, and who content themselves by saying in cases of death by Convulsion that the patient "died of an epilectic fit," and make no post-mortem examination, will not be aware that this diagnosis is sometimes grossly wrong.

In what follows, in order to encounter fully the difficulties of diagnosis which actually do occur, and because I have only to do with the most commonly occurring seizures (epileptic) in diagnosis, I will suppose that we are called to a person in his first fit, or first series of fits, and only incidentally notice what bearing on our diagnosis the fact of the previous occurrence of fits has. There is no position more embarrassing than that we are in when called to a patient in his first fit. As before said, it is easy to be *generally* right.

We will consider some of the recognised causes of Convulsion, or, it may be safer to say, the known conditions under which they arise. It will be well first to remark however, that when we are called to a patient who has "died in a fit," we must ask if the patient were eating when the fit came on. Dr. Lalor has written a valuable monograph on death by choking in epileptic attacks, and I could relate a case in which I feel convinced that death was thus caused, although the larynx was not examined *post mortem*.

Renal Disease (*Uræmia*).—In all cases of Convulsion we must examine the urine, however young the patient may be, and however healthy he may look, and notwithstanding that he has had fits described as epileptic on previous occasions. This examination is still necessary when the patient has recovered consciousness by the time we reach him. None of the above circumstances negative uræmia, nor is the quick repetition of fits of value in diagnosis. The fact that the patient has had no dropsy does not influence us. We must, I repeat, examine the urine. If it be smoky; if there be scarlet fever in other members of the patient's families; above all if the patient be the subject of scarlet fever, we conclude almost with certainty that there is uræmia. I say almost, because now and then Convulsions in scarlet fever are followed by a liability to Convulsions for life. As we sometimes say, "some cases of epilepsy date from scarlet fever;" occasionally they leave persistent hemiplegia. It is hard to believe that there can have been uræmia only in these cases. Since endocarditis occurs now and then in scarlet fever, and since plugging of the middle cerebral artery will cause Convulsions, it is as likely that there is embolism as uræmia.

Cerebral Hæmorrhage.—The mere presence of albumen, however, does not lead us to declare that there is uræmia. In a patient past middle age there may be cerebral hæmorrhage. If there be hemiplegia with deep and continuing coma, we diagnose hæmorrhage, and we do this notwithstanding that the patient is young—say twenty—and notwithstanding that he was "quite well before the fit." If there be no hemiplegia, and if the patient be young, the inference is very strong that there is uræmia, and not clot. But, as will be mentioned in the article on Apoplexy, a general convulsion, followed by deep coma and universal powerlessness, in a patient whose urine is albuminous, may be owing either to very *large* hæmorrhage into the cerebrum, into the lateral ventricles, or into the pons Varolii, or it may be owing to uræmia. We cannot rely on stertor, kind of coma, repetition of convulsion, or increase of temperature, although rapidly *increasing* stertor, rapidly *deepening coma*, are signs in favour of the diagnosis of hæmorrhage. It is the *ingravesence* of these symptoms, after the convulsion, which favours hæmorrhage. We will now suppose that there is no albuminuria.[1]

Cerebral Aneurism.—Dr. John W. Ogle and Dr. Murchison have pointed out that epilepsy (*i.e.* fits at intervals, like those usually called epileptic) occurs in patients the subjects of aneurism of large cerebral arteries. Such aneurisms will occur in young people, and therefore the question of age has no bearing on diagnosis. There is nothing special, so far as has yet been determined, in the kind of convulsion; there are, indeed, no symptoms which are characteristic of cerebral aneurism. There may be no symptoms at all, or none sufficient to send the patient to a doctor, until the fatal ones from rupture of the aneurism. We cannot therefore be certain whether a patient's fits are the results of cerebral aneurism or not. If, excluding albuminuria, syphilis, and other causes to be afterwards mentioned, we have reason to believe that there are vegetations on the heart's valves, we may surmise that the fits are owing to cerebral aneurism or (*vide supra*) to local embolism in connexion therewith.[1]

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[1] Occasionally when there is chronic renal disease, we discover no albumen in the urine, and it is said that occasionally, after a severe convulsion, albumen appears in the urine in consequence of that convulsion. I do not see how we can avoid mistakes in, these cases.

We can occasionally diagnose that a fatal seizure is owing to rupture of a cerebral aneurism, and this is an important matter in a medico-legal point of view. If a young patient has had Convulsions now and then for months or years, and if after one severe fit he is more profoundly comatose than usual, with great stertor, and if he continues so for some hours, rupture of a cerebral aneurism is probable. It is all the more likely if the patient *has* been hemiplegic. If the patient dies in the fit in a few minutes or in half an hour, we are more certain, because we know that it requires a large and, what is more important, a sudden hæmorrhage to kill quickly.[2] We are more sure still if the patient is known to be the subject of valvular disease of the heart, or if he has had rheumatic fever. Dr. J. W. Ogle and Dr. Church have shown, and my experience bears out their conclusions, that aneurism of the larger cerebral vessels frequently occurs along with vegetations on the heart's valves.

Embolism.—Embolism of the middle cerebral artery sometimes, although rarely, produces severe convulsion. It is followed by hemiplegia. The modes of onset of symptoms from plugging vary much. They sometimes come on suddenly and sometimes deliberately, according, the presumption is, as the vessel is slowly or suddenly plugged. Again, the degree of the symptoms varies. There may be no loss of consciousness, and the hemiplegia may be transitory. This is so when the branch occluded is small. If a patient the subject of valvular disease becomes hemiplegic after a severe convulsion, it is considered to be almost certain that there is sudden plugging of the main trunk or of a large branch of the middle cerebral artery: it is not quite certain. The convulsion and consequent hemiplegia may be owing to rupture of a large aneurism of this vessel. Aneurism of the middle cerebral artery usually ruptures so that the blood is poured out external to the brain, and the patient dies quickly because the blood gets out in large quantity, and, what is more important, with great rapidity. But occasionally it ruptures so as to *break up the motor tract*, corpus striatum, or thalamus, and will then, so to speak, imitate common cerebral hæmorrhage. If then a paitent, especially a young patient whom we know to be the subject of valvular disease of the heart, becomes hemiplegic after a convulsion, we must take this rarer possibility into consideration.

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[1] See Dr. Gull on Cerebral Aneurisms, Guy's Hosp. Rep. vol. v. (3rd series).

[2] It is not, of course, said that rupture of aneurism of the large cerebral vessels always kills suddenly or quickly, nor always by Convulsion. Rupture leads to death slowly, if the rupture of the aneurism is small, or if the blood, as when the aneurism s far in the Sylvian fissure, can only get out slowly.

If the coma be very deep, if it deepens, or, generally speaking, if the patient quickly gets worse, rupture of an aneurism is at least as likely as embolism. With all our care we shall be wrong now and then, as patients sometimes die in a few days in an apoplectic manner, from plugging of the middle cerebral artery. (See also Art. Softening.)

Tumours.—If the patient, especially if he be a young and healthy-looking man, have had for weeks or months severe pain in the head, vomiting, &c., there being no albuminuria,—above all, if there be also double optic neuritis,—a tumour of the brain is probable. Although headache is one of the symptoms of cerebral aneurism, cerebral aneurisms rarely if ever cause intense and persistent headache.

Here I must refer to the evidence stated in more detail (p. 287). But in this connexion one further fact is to be mentioned, viz. that a patient may have occasional convulsions for weeks or months from the "irritation of a tumour," and may die after one severe convulsion, or several quickly recurring convulsions, due to large hæmorrhage from that tumour. If, then, we find a patient whom we infer to be the subject of cerebral tumour who has been seized with convulsions much more severe than usual, and if he becomes unusually deeply comatose, and if the coma deepens, especially if there be no further convulsion, we fear large hæmorrhage from the tumour.[1]

Syphilis.—As before said, if the convulsion be of the first class, and if there be signs of organic disease, there is usually syphiloma of the brain; but if the convulsion be general, or if we know nothing of its mode of onset, we must infer from the evidence of present syphilis. If there be such demonstrative evidence as nodes, &c., or a clear history of recent syphilitic changes in any part of the body, our diagnosis is practically certain. If there is not such evidence, we may judge from the history of a random succession of nervous symptoms, such as palsy of a cranial nerve, followed by hemiplegia or paraplegia, or from the previous disorderly association of nervous symptoms, showing several lesions, *e.g.* palsy of the third or fifth, or portio dura with hemiplegia of the *same* side. When, however, such symptoms are of recent date, they may be still rarely owing to tumour. We must, however, always in these cases treat for syphilis.

Abscess.—Again, it is possible that there may be cerebral abscess. If we are to ignore this possibility altogether, we shall very rarely err, as cerebral abscess is very rare. I have, however, more than once been consulted for Convulsion which turned out to be owing to cerebral abscess. There are no certain points in diagnosis except the presence of bone disease in some part of the cranial wall, most often the bones of the ear, occasionally at the vertex. If the only evidence there is be that the patient a week or month ago received a severe blow on the head, the fit may be owing to *syphilitic* disease of the surface of the brain. It probably is, if the fit begins deliberately, and if there be epileptic hemiplegia. Syphilitic disease of the brain not infrequently follows blows on the head. If, however, there be no evidence of syphilis, no palsy of any cranial nerve—excepting amaurosis from double optic neuritis, which is scarcely to be called *palsy* of a cranial nerve,—if we find that there is a "puffy" tumour on the scalp, abscess is probable, and very probable if there be hemiplegia after the Convulsions. Occasionally, as is well known, a patient suffers from cerebral abscess without any symptoms at all, or any obvious symptoms. Occasionally after a period of latency it breaks into the lateral ventricle: then the symptoms are quite like those of hæmorrhage into the lateral ventricles, and we can only make the diagnosis of what has occurred from evidence of blows, disease of the bones of the ear, of the nose, &c. If there be no history, and if we find no evidence of disease of bone, we cannot make a diagnosis.

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[1] Of course it is not to be implied that hæmorrhage from cerebral tumour necessarily leads to Convulsions, any more than ordinary cerebral hæmorrhage does.

Epilepsy.—Supposing now that we can negative the above causes, we conclude that the patient has had an epileptic fit. By this we mean that he has had a convulsion which does not depend on an organic lesion, or on an acute state like uræmia, or on a sudden quasi-accident like hæmorrhage. We infer that he will quickly get into his usual health, but that he will in all probability have fits of a like kind again and again for years. We say probably, because now and then he does not suffer again, and it not infrequently happens that after the first fit or the first series of fits he has an interval of good health for many months.

DEATH IN CONVULSIONS.

Now and then, however, a patient dies in a convulsion, and we discover nothing *post mortem* which we can suppose to have been the cause of the fit. Of course there is something overlooked, and we should always search every organ of the body with great care in these cases. When the patient is known to have had fits of a like kind before, we may say that he died in an epileptic fit; but when it is his first fit, this nomenclature does not conceal the bald fact that a patient seemingly healthy has a convulsion the cause of which we cannot make out, even after post-mortem examination. He dies in it, we surmise, because it has been unusually severe, respiration, and probably the heart's action, having been suspended too long for recovery. The point that chiefly concerns us here is that such modes of death are well recognised, and do not indicate either violence or the administration of poison.

TREATMENT OF CONVULSIONS IN ADULTS.

Obviously enough, treatment will vary so much in different cases that most of what has to be said will be found in the articles Uræmia, Cerebral Hæmorrhage, Embolism, and Epilepsy. Indeed, I wish only to say a word on the treatment of Convulsions due to syphilis. In these cases we treat for syphilis, but in chronic cases, at least, this treatment is not of so great service, so far as removing the symptoms goes, as from superficial considerations we should expect. The bromide of potassium is of more service than the iodide in keeping off fits.

EPILEPSY.

By J. Russell Reynolds, M.D. F.R.S.

Definition.—Epilepsy is a chronic disease of which the characteristic symptom is a sudden trouble or loss of consciousness. this change being occasional and temporary, sometimes unattended by any evident muscular contraction, sometimes accompanied by partial spasm, and sometimes by general convulsion.

The two elements probably present in every case of Epilepsy are diminution of intelligence and excess of muscular contraction; and these two elements may exist in almost every variety of combination, and be developed to any degree of intensity. The latter element is not always seen to exist; there may be no spasm of the facial muscles, not the slightest change in the expression of countenance; or the face may become dull in aspect, or pale in colour, but consciousness is, for the moment, in absolute abeyance. There are reasons for thinking, as will be shown hereafter, that this loss of consciousness depends upon spasm affecting the vessels of the pia mater, but such spasm is hidden from our eyes. The former element, loss of consciousness, is that which is essential to our idea of Epilepsy; without its occurrence, no convulsion, however severe, should be regarded as epileptic; when it does occur, as a paroxysmal event, and with a chronic history, the case is one of Epilepsy, although no other symptom may be present.

There are two classes of errors into which authors have fallen with regard to the use of the word Epilepsy. The older mistake was to apply the term to every case in which there were convulsions, appearing in a certain form, called "epileptic," "epileptoid," or "epileptiform;" the modern error is to use the word to denote a paroxysmal—*i.e.* occasional and sudden—loss or dimunition not only of consciousness but of any function of any organ; or, indeed, sometimes to denote anything, or any condition, which occurs in a paroxysmal manner. The former led to the association, under one name, of diseases differing so widely from each other as tumour of the brain, Bright's disease of the kidney, intestinal entozoa, lead poisoning, and almost every form of malady: the latter might lead to the placing in one common group, and calling by one common name, such diseases as amaurosis from dyspepsia, stammering, deafness, paralysis, or asthma. The former tendency led to the production of such words as renal epilepsy, symptomatic and sympathetic epilepsy, toxæmic epilepsy, and the like: the latter has conduced to the coinage of such terms as epilepsy of the retina, acoustic epilepsy, and so forth.

There is, I think, a radical and very mischievous mistake in both of these modes of using words; the error is similar in the two, as far as regards its principle, but it differs in the detail of its development. The older authors exaggerated the importance of the form of a group of symptoms—convulsive—occurring in a number of organs, and common to many widely different diseases: the modern have exalted into undue prominence the pathological significance of one element out of this group of symptoms,—viz. arrest of function,—which single element may occur in many diverse organs of the body. By such a term as "renal epilepsy" was meant a disease resembling Epilepsy in its outward form, but dependent upon, not an unhealthy condition of the nervous centres, but on an irritation of the kidney, or an altered blood-state which kidney-disease might have determined: by such words as "retinal epilepsy" something very different is intended, viz. a malady showing itself only in the retina, in which a change takes place, supposed to be analogous, in its intimate pathology, to that occurring in the brain in Epilepsy. In the one Epilepsy merely means convulsion; in the other it merely means arrest of function; and the objection I entertain to such use of terms is based upon the fact that, however widely different individual cases of Epilepsy may be, they do yet belong to and constitute a group which has a definite clinical history, and has had it for some hundreds of years. If good reason can be shown for getting rid of the word "epilepsy," I should rejoice to lose it from our nosology; but so long as the word is retained at all it should have a definite and intelligible meaning. Renal asthma would be a term as pathologically correct as "renal epilepsy;" dyspnœa of the fingers as justifiable as the expression "epilepsy of the retina."

Synonyms.—No useful end would be served by enumerating all the names by which this disease has been described, inasmuch as many of them have fallen into complete disuse. The most important are the following:—

Epilepsy (English); l'Epilepsie (French); Fallsucht (German); Mal Caduco (Italian); Epileptica passio, Morbus sacer, M. comitialis (Latin); ’Επιληψία, ’Επιλῆψις (Greek).

Natural History.—1. General Prevalence of the Disease.—Epilepsy is spoken of as a very common affection. Niemeyer states that in every thousand individuals there are to be found six epileptics.[1] Such statement cannot, I think, be true with regard to Epilepsy in this country; for among 1,820 invalids, whose cases were recorded by myself as out-patients of the Westminster Hospital, there were only seven epileptics; and but thirty-four whose diseases could by any possibility be confounded with Epilepsy. It must be observed further, that Niemeyer is speaking of individuals generally, and that the results of my own examination at the Westminster Hospital are obtained from a small class of individuals, viz. those who are ill.

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[1] Niemeyer, Handbuch der speciellen Pathologie, p. 637.

The proportion of true Epilepsy to other diseases of the nervous system has been found to be about 7 per cent.

2. CAUSES OF EPILEPSY.—(*a*) *Predisposing Causes.—Hereditary taint* has been found to exist in rather less than one-third of those cases which have fallen under my care, and have been carefully examined on this point.[1] It is not intended by this statement to affirm that true Epilepsy has existed in the parents of one-third of the cases; but that some disease of the nervous system, more or less closely allied to that under consideration, has been present in either the parents, the grandparents, the aunts, uncles, brothers, or sisters; that there has been a family proclivity to nervous disorder, in one case showing itself by idiotcy, in another by mania, in a third by convulsions, and so forth. I have found only 12 per cent. of epileptics giving a distinct history of Epilepsy in other members of their families; a number which is very near to that stated by Dr. Sieveking, and not far removed from that given by M. Delasiauve.

It has been said that the disease is more frequently transmitted on the fathers' than on the mothers' side,[2] but the reverse of this proposition has been found to obtain in cases examined by myself.

Of 130 epileptics, I found 80, or 61·06 per cent., who asserted the entire absence from their families of any predisposition to nervous disease; and 8 individuals, or 6·10 per cent., who were in some uncertainty as to the health of important relatives. These patients were derived from all classes of society; and I have no means of determining the question, on a scale sufficiently large to be satisfactory, whether Epilepsy is more commonly found to be hereditary in the upper, the middle, or the lower classes. Several elements of doubt enter into the solution of this question, the most important of which is the greater difficulty that is encountered in obtaining accurately the facts which belong to the latter. Hospital patients often know but little of their antecedent or even collateral relations. Among the upper classes there is not rarely a studious concealment of what are regarded as prejudicial family conditions. The middle classes are not only more accurate than the former, but more free than the latter; and, judging from what I have gathered from them, as they shade off on either side,—above them and below,—I should be of opinion that hereditary taint is more frequently discoverable in the better conditions of life

[1] In a careful paper by Messrs. Leech and Fox, in vol. i. of the Manchester Medical and Surgical Reports, p. 198, the proportion of those epileptics in whom hereditary taint was traceable was somewhat higher, viz. 36·8 per cent. These observers compare with each other epileptics and non-epileptics, and, having obtained particulars with regard to the health of a large number of the relatives of each group, show that the relatives of epileptic patients were found to suffer from "some form of nervous disease" in larger proportion than were those of non-epileptic individuals.

[2] Esquirol. Des Maladies Mentales, tom. i. p. 406.

than in the poorer. It is not intended that there are absolutely a larger number of hereditary epileptics among the former than the latter; but that, of an equal number of epileptics in the two extremes of society, a larger proportion will furnish evidence of hereditary taint among the rich than among those who are in want. This is probably due to the fact that the latter class are exposed more frequently and more severely than are the former to the most active determining causes of the disease, viz. anxiety, alarm, and want. With regard to the hereditary transmission of Epilepsy, as indeed with regard to the causation of all diseases by supposed hereditary taint, it must be remembered that, inasmuch as the large majority of cases owe their malady to other causes than inherited tendency, a certain number of those whose parents exhibit a like affection to their own may have become morbid independently of any hereditary taint. It is well known that many of the children of epileptic parentage are free from the disease, and it is quite clear that many epileptics, descended from epileptic stock, have been exposed to causes of the malady which would, of themselves, have been held sufficient to have induced the malady independently of any constitutional taint. It is, therefore, of practical importance not to assume too readily the operation of this cause, and hence to neglect an examination into other conditions. In the largest and most correct sense of the word, the etiology of Epilepsy is advanced but little by the discovery of hereditary taint; the causation may be thus thrown backwards, but it is not explained.

Sex.—Little that is of value can be shown with regard to the influence of sex as a predisposing cause of Epilepsy. Practically, the two sexes appear to be about equally affected; and the different statements that have been made by various authors—some of whom represent the male sex, others the female sex, as the more liable to the disease—may probably be accounted for by other circumstances than that supposed, viz. a special sexual predisposition. The relative number of female epileptics who are out-patients of hospitals may be determined by the hours at which the physicians make their visits, or by other conditions which have to do with the social position of the applicants, and which may render it easy, difficult, or almost impossible for either the one or the other sex to attend.

Similar degrees of fallacy, although different in kind, may influence the results obtained from private practice. The facts of a physician's age, and single or married condition, for example, might exert an influence upon the relative numbers of his male and female patients too great to be counterbalanced by proclivity to Epilepsy inherent in either sex.

Again, the statistics gathered from asylums are liable to disturbing causes so far as etiology is concerned. In proportion to the amount of disease a larger number of males than females find their way into public asylums. The reason for this is obvious, viz. that men are prevented from doing their special work in the world by an amount of disease which need not deter women from performing their domestic duties. Yet further, the statistics furnished on this point by some authors are complicated by limitations as to age, and by the fact of more or less clearly pronounced insanity of mind.

Little, then, that is definite can be stated on the influence of sex, as a predisponent to Epilepsy; and it seems to me to be the wisest course at present to leave the question open for further investigation.

Age.—The influence of age in the production of Epilepsy is strongly marked. This is shown in the following short table of cases collected by myself:—

Age at commencement.	Males.	Females.	Total.
Under 10 years	10	9	19
Between 10 and 20 years	66	40	106
Between 20 and 45 years	25	20	45
Over 45 years	1	1	2
	102	70	172

The most important fact to be recognised in the above summary is the great frequency with which Epilepsy commences between 10 and 20 years of age—*i.e.* at a period of life embracing the processes of the second dentition and of the establishment of puberty; and, without going much further into detail, it may be stated in addition, that by far the larger number of the group showed their first symptoms of the disease between the ages of 13 and 17 years, inclusive. Further, that there is a comparative immunity from the commencement of the disease between 25 and 35, the greater proportion of cases forming the third group having been seized by the disease at or about the age of 40.[1]

When there is a marked hereditary taint as a predisposing cause of Epilepsy, the disease is found to develop itself somewhat earlier than under other circumstances. The difference, however, is not so great as that which is to be observed in regard of some other maladies which are held to be hereditary. The difference may be fairly represented in the following table:—

Commencing under æt. 15 . . .	83·33 hereditary	46·15 non-hereditary.
above ,, . . .	16·66 ,,	53·82 ,,

It has appeared, further, that when Epilepsy is hereditary it shows itself at an earlier age among girls than among boys. The difference is not great, and Messrs. Leech and Fox have arrived at an opposite result.[2]

(*b*) *Accidental or Exciting Causes.*—Patients and their friends often exhibit a very great anxiety to refer the outbreak of Epilepsy to some external condition, which they may speak of as its cause; and, in doing so, they occasionally attach undue importance to trivial circumstances. There is a natural reluctance to admit the presence of constitutional or hereditary taint, and an eagerness to find excuses for the poor sufferer, in the fact of his having been exposed to some extraordinary disturbance from without. In this way we may in some measure account for the wideness of the range of conditions to which the production of Epilepsy has been referred. It is so difficult to conceive that a disease having such strongly marked features as those of the epileptic paroxysm can lurk in an apparently healthy frame—that all the essential conditions of so terrible a malady may be present and yet give no sign—that many find an explanation of the outburst in some externally disturbing cause which they can appreciate, and ignore the operation of those internal conditions which had hitherto escaped their notice, or had been regarded from a different point of view.

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[1] For further information on these points the reader is referred to Hasse, in Virchow's Handbuch, 1ster Abth. 4ter Bd. p. 264; Reynolds on Epilepsy, p. 126; Leuret, Archiv. Gén. de Méd. 4me Série, 1843, t. ii.; Sieveking, Med.-Chir. Trans. vol. xl. p. 158; Herpin, Du Pronostic, &c. p. 332; Leech and Fox, in Manchester Medical and Surgical Reports, p. 199. It is, however, to be remembered that in the case of some of the authors referred to care has not been taken to separate Epilepsy from other convulsive diseases.

[2] Op. cit. p. 202.

It is important to classify the causes to which Epilepsy has been referred, and I have done so by distributing them into four groups; placing in the *first*, those which operated through the mind or the emotions, such as fright, grief, worry, and the like; in the *second*, those which acted through the reflective centre, such as eccentric irritations; in the *third*, those which produced their effect through changes in the general health, such as those which may be occasioned by pregnancy, by acute specific or other diseases; and in the *fourth*, those which may be regarded as acting physically, such as insolation, mechanical injury, and the like. It is difficult to determine into which category of causation some cases should be placed; as for example those in which the fits have been referred to either falls or blows, inasmuch as it is possible that such accidents may have operated through the mind by alarm or fear, rather than through the body by the merely physical process of concussion or laceration. I have placed such cases in those groups to which they had been assigned by the patients or their friends at the time that the disease began.

The following table exhibits the relative frequency of the several kinds of causes to which I have referred:—

Nature of Cause.	Number of Cases.
I.—Psychical; such as fright, grief, worry, over-work	29
II.—Eccentric irritation; dentition, indigestion, venereal excesses, dysentery, &c.	16
III.—General organic changes; fatigue, pregnancy, miscarriages, rheumatic fever, scarlet fever, diphtheria, pneumonia	9
IV.—Physical influences; blows on head, falls, insolation, cuts	9[1]
	63

Besides these sixty-three cases, I have the records of sixty-one cases in which no cause could be assigned; the patients or their friends either asserting their absolute inability to make any reasonable conjecture on the matter, or hazarding some explanation which was utterly nonsensical. It is important to know that of these sixty-one, there were forty-three individuals who, after examination and cross-examination, and suggestion, could give neither to themselves nor to me any clue to the solution of the mystery. Of 124 cases, therefore, sixty-three, rather more than the half, supposed that they could explain the causation of their malady; forty-three, or 34 per cent., asserted their utter inability to do so; while twenty-nine, or 23 per cent., referred their attacks to mental or emotional disturbance.

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[1] Messrs. Leech and Fox found a much larger proportion of cases falling under this category. Op. cit. p. 206.

The frequency with which mental or emotional disturbance has been shown to be the cause of Epilepsy is such that it requires some further notice. The most common conditions that I have witnessed are those of continued anxiety and prolonged rather than intense alarm. I have in a very few instances found that an over-strain of the mental powers has been followed by Epilepsy, but in almost every one of these cases there has been considerable anxiety as well, and it, I believe, has been the more efficient factor of the malady. Women and girls have much more frequently than have either men or boys referred their attacks to emotional disturbances; the proportion being 36 per cent. of females, and 13 per cent. of males. The period at which the first attack has occurred after an individual has received some great mental shock varies widely; the fit may take place at the moment of alarm, or it may follow after an interval of hours, days, or weeks.

With regard to eccentric irritations, it must be remembered that in the list given above cases of "convulsions" are not enumerated. Both the first and the second dentition, and even the cutting of the "wisdom teeth," may be attended by convulsions, which in the large majority of cases disappear as soon as the source of annoyance has been removed. In a few rare cases, however, the processes referred to have appeared to cause genuine Epilepsy, and it is to these rare cases that reference is made. It is curious to know that in not more than half of the cases of Epilepsy can it be ascertained that "fits" have occurred during infancy; and it is a still more interesting fact that epileptic women appear to exhibit no high degree of proclivity to puerperal convulsions. Dr. Tyler Smith states that puerperal convulsions occurred only twice in fifty-three deliveries of fifteen epileptic women;[1] and so far as my own experience extends, it is exceedingly rare, and indeed almost unknown, for epileptic women to suffer from their attacks during or immediately after labour.

Among the second group of causes appears one to which I believe, far too great an amount of importance has been attached, viz. excessive venery or masturbation. It is very common to hear suspicions expressed upon this point; much more common, I think, than to hear any such statement of facts as should prove that Epilepsy and masturbation have any special character or frequency of relation to one another. The one is a tolerably prevalent disease, the other a very widely distributed vice. There are multitudes of epileptics with regard to whom no such suspicion could ever be entertained; and there are, it is to be feared, much larger multitudes of masturbators who have never become epileptic. When, therefore, we find the two elements combined in the same individual, it is necessary to observe some caution in our attempt to interpret their relations. It is, I believe, sufficiently well proved to be regarded as a fact that the vice referred to is liable to induce various disturbances in the health, and that the major part of these are brought about by, and are exhibited in, the altered functions of the nervous system; but what it appears to me is yet wanting in proof is the special relationship of Epilepsy to this particular wickedness or weakness. Again and again it has occurred to me to see cases of vague and various nervous derangements which might be fairly inferred to be the result of masturbation; but it has in only an exceedingly small number of cases of Epilepsy been possible for me to establish the existence of such relation.

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[1] Lancet, 1849, vol. xxiv. p. 644.

There can, I think, be no doubt whatever as to the existence of an intimate association between various forms of nervous malady and either various abnormal conditions of the sexual organs, or unnatural circumstances attending upon their exercise; but, as yet, the nature of that association is, I believe, and as undoubtedly, unexplained. Sometimes sexual excess, and sometimes the reverse; now great emotional involvement, and now the entire absence of all sympathy; at one time exuberant enjoyment, and at another disappointment or disgust, are conditions met with in epileptics, and in all forms of many sorts of disease; but, so far as I know, neither one of those conditions is more frequent than another in the history of epileptics. I have known cases in which morbid libidinousness occurred in epileptics, but only long after the development of the disease; and on the other hand I have met with cases where the sexual propensity had become diminished, or even extinct, after the occurrence of the attacks, and this without any previous excess in its gratification.

In endeavouring to determine this question, which is of considerable etiological interest, it would be undesirable to omit notice of the striking effects which have been observed to follow the administration of bromide of potassium in cases of Epilepsy. It cannot be doubted that this medicine is highly valuable in diminishing the numbers of attacks;[1] and the only point of interest to us now is to ascertain whether its *modus operandi* is such that it either countenances or discountenances the prevalent belief with regard to the etiological question under consideration. When this medicine was first introduced by Sir Charles Locock,[2] it was recommended as being of especial service in those cases of Epilepsy in women in which the attacks occurred only during the menstrual period; and since that time it has been very generally received that bromide of potassium possesses strong antaphrodisiac properties, and that its utility in Epilepsy is to be accounted for by its special action upon the generative organs. From the very first I saw reason to doubt this mode of explanation,[1] and much enlarged experience has, from my own mind, removed all doubt whatever upon the point, and produced a settled conviction that bromide of potassium, when given in such doses as to be of service in Epilepsy—viz. from 10 to 30 grains either three or four times daily—exerts no recognisable influence upon either the sexual propensity or power. It is not asserted that doses might not be given so large as to exert such influence, but that where decidedly remedial effects have been produced in Epilepsy, their production has not been attended by any change in the generative functions. Dr. Duckworth Williams[2] says: "I have tried it (K Br.) in every variety of uterine affection that has come within my reach, including nymphomania, satyriasis, menorrhagia, amenorrhœa, dysmenorrhœa, &c. &c., but without perceiving the least benefit accrue." Dr. Williams mentions cases in which the patients, in spite of their taking the medicine, "persisted in their bad habits, and their sensuality became if possible more confirmed" (p. 17); and his experience on this matter is in entire accordance with the results of my own observations. We cannot, therefore, support the prevalent creed in regard of one mode in which Epilepsy is produced by facts gathered from the treatment of that disease by bromide of potassium.

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[1] See p. 321 on Treatment, Dr. S. W. Duckworth Williams' Paper "On the Efficacy of the Bromide of Potassium in Epilepsy and certain Psychical Affections," also a paper by the editor in "The Practitioner."

[2] See Lancet, May 20, 1857, vol. i. p. 528.

To what degree the view to which I refer is supported by the observations of Mr. Baker Brown,[3] must depend partly upon the therapeutic results of his mode of treatment, and partly upon the interpretation which must be given to the alleged facts. On the former point the evidence is unsatisfactory, being gathered from a small and too exclusive selection of cases;[4] on the latter point some misconception is possible. Considered *etiologically*, we want to know the proportion of cases in which the particular cause to which Mr. Brown refers had been in operation, but upon this point we are not furnished with any evidence whatever, inasmuch as in *all* the cases he records not only was irritation of the pudendal nerves believed to exist, but a certain kind of operation was performed. It would, I think, be pushing much too far the inference to be drawn from Mr. Brown's little book, to assert that his opinion is that *every* case of Epilepsy is produced in the manner described. What we want to know is the number of cases of Epilepsy in which Mr. Brown entertained no such suspicion, and, still further, the number of cases in which, having entertained it and acted upon it, the result was unsatisfactory. As to the interpretation of the facts that are stated, there is this to be borne in mind, that so far as I can understand Mr. Brown's theory, it is not that in such cases there have been of necessity immodest wishes, excessive sensuality, or irregular practices, but that there has been a morbid condition of irritability of a certain nerve, and that this has been taken away by the removal of the peripheral termination of the nerve. Referring for future consideration the question of the therapeutic propriety or desirability of the operation of clitoridectomy,[1] all that it is necessary to say now is that—in the absence of any definite statement of Mr. Brown upon the question of proportion as described above—my own experience would lead me to believe that the cause he refers to is of very rare and very exceptional occurrence.

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[1] See Author, on Epilepsy, p. 332.

[2] Loc. ant. cit. p. 16.

[3] On certain Forms of Insanity, Epilepsy, Catalepsy, and Hysteria in Females.

[4] See Treatment, p. 324.

In another work [2] I took some pains to show how extremely rare it was to meet with a case of Epilepsy in which no causative conditions could be discovered. Although in one person we might find no predisposing cause, and in another no exciting cause, in only one-eighth of the cases was there an absence of both. In seven-eighths, either one, two, three, or more causative conditions of disturbance were present and were recognised.

The proportion, therefore, of cases of Epilepsy in which the causation of the disease is placed beyond explanation by our present knowledge of pathology is not greater than that which we meet with in many other chronic diseases, and is far less than that which is admitted to exist in several. There is some mystery in the causation of almost all diseases; I do not think that it is greater in the case of Epilepsy than in that of many others with regard to which we think ourselves on easy terms with the science of pathology.

3. SYMPTOMS.—It will be convenient to consider those which occur in, and constitute a paroxysm of Epilepsy, separately from such as may be observed during the intervals of attack. We have, therefore, to describe, first—

The *Paroxysmal Symptoms*, or features of the *attack*. In the most characteristic cases of Epilepsy there is an entire loss of consciousness in conjunction with a peculiar series of involuntary muscular movements; but, on the one side of these typical cases, we see epileptics in whom the loss of consciousness is alone obvious, and, on the other, individuals exhibiting certain highly marked spasmodic phenomena, and only very slight or even imperceptible obscuration of the mind. It is necessary, therefore, to classify cases, in order to render description possible, and it is proposed to do so by dividing them into four groups, which may be thus distinguished :—First, those in which there is loss of consciousness without evident spasm; second, those in which such loss of consciousness is accompanied by local spasmodic movement; third, those in which it is attended by general tonic and clonic convulsion, following a particular order; and fourth, those in which general or partial convulsion occurs without complete loss of consciousness. The first and second forms may be termed "epilepsia mitior," or "le petit mal;" the third form "epilepsia gravior," or "le haut mal;" and the fourth "epilepsia abortiva," or irregular Epilepsy.

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[1] See p. 324.

[2] Author, on Epilepsy, p. 261.

(*a*) *Epilepsia Mitior, or "Le Petit Mal," without evident Spasm.*—All that occurs and can be positively attested in cases of this description is a sudden, temporary, but absolute arrest of both perception and volition. The individual so attacked loses consciousness for two, three, or more seconds; and may after that or a longer period resume his sentence or employment, perfectly unaware that anything abnormal has happened.

Sometimes there is slight loss of balance—the patient, if standing or walking, leans to one side, or staggers, but does not fall; sometimes there is pallor of the countenance followed by slight flushing; sometimes the latter without the former; sometimes there is slight dilatation of the pupil, and an absence of the expression of "looking at anything;" sometimes an irregularity and faltering of the pulse; but often, as I can testify from repeated observations, there is not any one of the physical changes I have mentioned; the patient's mind becomes a blank for a few seconds, and that is all that can be observed.

These seizures are often regarded as "faintings," and are described by patients under various terms, such as "blanks," "forgets," "faints," "sensations," "absences," "darknesses," &c. &c.

Occasionally these slight attacks are preceded by vertigo; the patient thinks that he shall fall, and so lies down to avoid doing so: sometimes he staggers and grasps an object for support; but, much more commonly, he simply ceases to perform any act requiring volition—he stops speaking or writing; but the automatic movements of standing or sitting, and the secondarily automatic movements of riding, walking, or holding an object, are maintained.

Sometimes the attack is followed, for a few seconds or for a longer period, by an obscured or altered state of intelligence; the patient speaks in reply to what is asked of him, but in half an hour afterwards is found to have entirely forgotten what was said to him or by him. In more rare cases the mind is dull, or altered from its habitual condition for a period of some hours, the patient being low-spirited, or suspicious, and apparently labouring under some delusion which he afterwards forgets. In this condition he may be listless; or he may do some odd things which he cannot afterwards account for or even recollect.

(*b*) *Epilepsia Mitior with evident Spasm.*—This is more common than the preceding, which it resembles exactly so far as the mental condition is concerned. The extent and locality of the spasm differ widely in different cases, and also in the same individual at different times. There may be only slight strabismus, or drawing of the mouth, partial turning of the head to one side, or some movement as of swallowing or attempts at getting something from the mouth; or, on the other hand, there may be slight momentary rigidity of the whole body. Sometimes the patient fixes his chest walls, and appears to "hold his breath;" sometimes he does some curious thing, such as stoop down to peep under a sofa, lie down and pull off his cravat, jump from his chair and walk quickly half-way across a room; but in any or all of these apparent attempts to do something he is suddenly arrested by the loss of consciousness, which is often absolute. Occasionally peculiar actions are performed after an attack of "le petit mal;" but it has never occurred to me to find an epileptic who could tell me why he did these things, or who could even remember that he had done them.

As to the locality of the muscles affected, it would appear that those of "expression" and of respiration are by far the most frequently involved. There is no evidence to show that either "trachelismus,"[1] or "laryngismus," or "phlebismus" occurs with anything like such frequency as to make them of any value in the interpretation of the epileptic paroxysm; although it is quite clear that the former may exist to such a degree as to occasion duskiness of the face.

The spasm in "le petit mal" is never violent; and it is only of short duration. It is tonic in its character, and painless to the patient, and the vascular changes which may be observed are of the same variable degree and kind as those enumerated in the previous section. Patients sometimes have warning sensations of these attacks, and I have known more than one instance in which there was a highly marked and most painful "aura epileptica."[2]

The most common combination and degree of symptoms may be thus described,—a feeling of giddiness, faintness, or discomfort; slight twisting of the neck, with anxious, lachrymose expression of the face, dilatation of the pupils, and pallor; accompanied, or quickly followed, by entire loss of consciousness, which lasts for two or three seconds; the patient "becoming himself again" after making a few sighing sounds, but feeling faint and bewildered, and often perspiring freely.

(c) *Epilepsia Gravior, or "Le Haut Mal."*—This, the ordinary form of Epilepsy, is in the vast majority of cases characterised by complete loss of consciousness, and a peculiar combination and series of spasmodic movements. In very rare instances we have the latter element without the former; the more common and much larger group shall be described first, and it will be convenient to enumerate separately the premonitory symptoms, those of the attack, and the immediate sequelæ or after-symptoms.

Premonitory symptoms are sometimes absent altogether; in certain cases they are of regular occurrence, being in the same individual invariable in character, while in another set of cases they are sometimes absent and sometimes present, and are more or less variable in their features. Their duration may be almost momentary, it may extend to several minutes, or, but very rarely, to hours, or even days. When of long duration, the prodromata are diminished in speciality and in intensity; and consist, so far as I have seen, in some mental change, or in some alteration of the general appearance. Thus, there may be an exaggeration of any habitual condition of the mind or spirits; the patient becoming, to an unusual degree, depressed, morose, or taciturn; or, on the other hand, lively, irritable, and excited. I have known several instances in which an undue flow of spirits, and an emphatic frivolity and expression of "feeling remarkably well," have almost invariably preceded the epileptic paroxysms. Such sensations have occurred in those patients whose attacks were not of very frequent repetition. It is very difficult to describe those changes in general appearance, or in "the looks" of a man, which friends recognise as premonitory of an attack. Generally, I believe, they depend upon an alteration in the colour of the skin, and some want of fineness in the outline of the features. The face becomes less red, more yellow, and somewhat dusky in tint, and there is a certain puffiness which, without altering in kind, diminishes in force its habitual expression. It is said, "He seems quite himself, but he does not look so; he is sharp enough, but looks stupid; and we know that an attack is coming on." There is no œdema, but there is a partial obliteration of the lines which make up "expression."

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[1] Marshall Hall. "Memoirs on the Neck as a Medical Region," 1849.
[2] See p. 304.

Those symptoms which *immediately* precede the seizures are widely different in character, variability, intensity, and duration. They may occur in the mind, the sensations, the muscular system, or the general bodily condition.

The mental prodromata are of many kinds: in some cases there is a distinct idea, never spontaneously presenting itself at any other period, and one which in its character and bearing is perfectly remembered afterwards; while in others there is a vague notion, recognised to be the warning of an attack, but of such indistinct character that only the fact of having entertained it is remembered. One gentleman told me that just as an attack was coming on he always thought, "This is what I had foreseen, I knew it would come on here, I ought to have avoided it by remaining away;" and this, although there had not been the remotest suspicion beforehand that an attack was imminent, or that the circumstances about to be entered upon would be likely to induce it. Much more common is a vague feeling of fear, which is horrible enough, but happily of only short duration.

Sensorial changes are by no means uncommon, and they are of every kind, description, and indescribability. By far the most common is a "painful feeling," sometimes said to be "most painful," or "horribly painful and distressing," but which, yet, the patient says—when minutely questioned—is not "pain," in the ordinary sense of the word. It would seem to be some condition of sensation which is intensely distressing, but which is unlike what we mean by smarting, burning, aching, &c. &c. Patients sometimes say that "it is in the head," and yet it is "not headache;" that it is in the epigastrium, and yet is not "stomach-ache." In some cases the sensation is always in either the head, the epigastrium, or lower thoracic region, the lower abdominal region, or the limbs. These are stated in the order of frequency, as they have occurred to me. In a large number of individuals, however, the sensation—which is sufficiently distinct and consistent for them to know that an attack is coming—is so vague that they cannot assert whether it is in the head, chest, abdomen, or limbs. Sometimes there are hallucinations of the special senses; one patient told me that he always heard "an infernal noise, something like that outside a booth at a country fair;" another that he had "a vision of a hideous donkey." It would be waste of space to enumerate further these prodromata.

Premonitory symptoms may occur in the form of tremor, twitching, tonic spasm, or co-ordinated movements, such as turning round, running some distance, &c.; or they may appear as partial paralysis of one or more limbs. The latter is stated to be more common in old people.[1]

The term epileptic "aura" has been sometimes used, very vaguely, to describe any premonitory symptom of which the patient could give an account; but, when more strictly limited in its meaning, it has been used to express a sensation of blowing, or of something analogous thereto, which, commencing in the periphery, passed upwards to the head, the patient becoming insensible when it had reached this point. Passing over for the present the pathological interest attaching to the interpretation of the so-called aura, it may be now stated broadly that anything characteristic of Epilepsy, in the second or limited sense of the word "aura," is rare, but that when such premonitory symptom does occur, it varies in character in different individuals; in one class there is a pain in the limbs, which "runs up them towards the head;" in another there are some twitching movements, and "the leg draws up," or "the arm becomes contracted;" and in a third there is some vague uneasiness about the hypogastric or epigastric regions, which "goes up through the chest." One peculiarity attaching to these symptoms is the facility with which they may sometimes be removed, and the attack averted. Pain may be stopped by rubbing, or by the pressure of the hand, or of a ligature; contractions may be undone by forcible extension of the limb; and the uneasiness in the abdomen may be removed by a cordial draught.[2] The duration of the aura is very variable, viz. from a few seconds to several minutes; sometimes the feelings "come, and go again," for hours, being arrested many times in the manners I have mentioned, but at last, as the patients say, "slipping by," and being followed by the fit.

There are, further, premonitory symptoms in the vascular system, and in the secreting organs, such as alterations in the colour of the face, or of the fingers, a redness or duskiness of the lips, a blue colour of the gums, an excessive salivation, a change in the nature of secretions;[3] but these are of rare occurrence, and of such variable character that the mere fact of their existence is all that need be stated here.

The relative frequency of the different classes of premonitory symptoms, so far as I have been able to ascertain, may be represented thus:—

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[1] Tissot, Traité de l'Épilepsie; Œuvres, tome vii. p. 131.
[2] See cases recorded by Author, op. cit. p. 92.
[3] Romberg, Manual, Syd. Soc. Transl. vol. i. p. 198.

Mental and emotional	11·1	per cent.
Sensational	19·8	,,
Motorial	8·6	,,
Vascular and secretory	3·7	,,

Prodromata were declared, positively, to be absent in 40·7 per cent.,[1] whereas information was "doubtful" in regard of 16 per cent. The most common precursory sensation was vertigo; there was little difference to be observed between the relative liability of the two sexes to any one form of "warning."

Actual Symptoms of the Attack.—For the purposes of description it is desirable to divide the epileptic paroxysms into three stages.

In the *first* stage of the attack there are the following phenomena, which occur—not successively, as they are necessarily represented in writing, but simultaneously, or with only slightly varying order:—

Loss of consciousness, *i.e.* of perception and volition.

Tonic contraction of the muscles throughout the body, with some excess of power on one side, or in one direction.

Impeded or arrested respiration, with or without a crying noise.

Pallor, redness, or duskiness of face; either the one or the other, often the one succeeding the other in the order they are mentioned.

Dilatation of the pupils of the eyes.

Natural, weak, or imperceptible radial pulses, with throbbing carotids and distending veins.

This stage lasts from two or three to thirty or forty seconds.

The loss of consciousness is usually sudden and complete; the patient falls down, or is, as it were, "thrown down" in a moment, with or without warning; but even when the warning occurs, so that he may change his position, or call attention to his wants, habitually the passage from consciousness to unconsciousness is abrupt, and the loss absolute. Sensation is, at the same time, in abeyance; although some reflex acts may be excited.

The tonic spasm of the muscles is peculiar, and it may precede the loss of consciousness. The patient usually appears to be straining round towards one side, as if striving to look over and behind one of his shoulders. The muscles of the face and front of the neck are those which most frequently mark the onset of the paroxysm.[2] The eyeballs, the head, the arms, and the trunk, turn and twist round, so as to give the impression I have mentioned. There is universal strain, but not actual equilibrium. Every limb is rigid, every muscle is at work; but some one set of muscles in each limb proves slightly stronger than its opposing set; and the limbs pass slowly, in a stiffened manner, and sometimes with slight jerking movements, from the positions that they occupied before the attack commenced. The head, neck, and trunk share in a similar movement, and its direction is usually uniform in the individual epileptic.

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[1] Messrs. Leech and Fox give a smaller proportion. Op. cit. p. 218.

[2] In twenty-four of forty-two cases. Leech and Fox, op. cit. p. 222.

Respiration is arrested, the patient appearing just like a man forcibly "holding his breath;" and in nearly half of the cases which have fallen under my own observation, the stoppage of the breathing has been so complete that no sound whatever has escaped from the mouth. In a certain number of individuals the respiration proceeds without actual interruption, but its movements are diminished in force; whereas in a very small number there is no change whatever.

In an uncertain proportion of cases there is the "epileptic cry," a peculiar and hideous sound, of which there are two distinct varieties. Some individuals utter a yell at the very commencement of the attack, and just before there is the peculiar holding of the breath I have described. Others do not "cry," but emit a groaning sound, which is, as it were, squeezed out of them by the quasi-tonic contraction of the muscles of the chest. There is, in fact, in regard of respiratory movement, a condition analogous to that observed in the limbs and trunk, viz. that of strain, but of imperfect equilibrium. As in the limbs there is a stiffened movement, from the fact that one set of muscles overcomes its opponents, so in the chest, sometimes a slow expiration, sometimes an inspiration is performed, and with either of these there may be a groaning sound. Usually there is but one sound—either a yell or a smothered groan; there is no repetition of either the one or the other.[1]

Pallor of the face is observed immediately before, and at the very onset of the attack in many; it is not present in all; and it occurs more certainly and more notably in females than in males. In other instances the face remains absolutely unchanged in regard of colour, whereas in a larger number there is suffusion of a florid, dull red, or dusky hue. Messrs. Leech and Fox found pallor to exist in only 38 per cent. of their cases, whereas duskiness was "very marked and present all through the fit" in 53 per cent.[2]

Dilatation of the pupil occurs, and, so far as I have seen, invariably, at the onset of attack. In one case, however, I witnessed a momentary contraction before dilatation commenced.

The pulse, as felt at the wrist, is usually small, and is sometimes quite imperceptible; but in several cases that I have observed there has been no change whatever in either the force or rapidity of its beats. When it has been imperceptible, there has been highly marked tonic spasm of the limbs; and often at the same time the heart may be seen, felt, and heard to be acting, and that even forcibly, and there is obvious throbbing of the carotid arteries.

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[1] I state this as the result of special attention to this point, as in a singular case, occurring many years ago, the question of the possible number of "cries" an epileptic might make assumed some importance in a medico-legal inquiry.

[2] Op. cit. p. 224.

In the *second* stage of the attack there are the following symptoms:—

Persistent unconsciousness.

Clonic convulsion.

Laborious breathing, with gurgling, foaming, and the like.

Darkness of face, and body generally, with cold and often profuse sweating.

Oscillation of the pupils.

Throbbing, laboured pulse, and palpitation of heart.

This second stage may last from a few seconds to five or ten minutes, its features gradually passing into those of the third stage. The transition from the first to the second stage is abrupt, and is determined by what may be termed the "letting go" of the breath which had been "held" before.

Clonic spasms are, more or less, universal; often they begin in the extremities, and are more highly marked on one side of the body than on the other. The jaws are champed together, the tongue is bitten, the limbs are thrown about, the bladder, rectum, and vesiculæ seminales may be evacuated; there are rumbling noises in the intestines, hiccup, and vomiting. The eyeballs are rolled outwards, and in every direction but that which is natural, and the aspect is as hideous as can be conceived.

Respiration is violently and convulsively performed; the diaphragm may be felt through the abdominal walls; the chest heaves; the alæ nasi are forcibly dilated; and the patient is in the condition of one who has made a most violent effort, and is now "out of breath." Mucus is heard rattling in the trachea, and is often blown out of the mouth, bloody from the bitten tongue or cheek. There is obviously great excess of secretion, and much of the distress of the sufferer appears due to his want of power to get rid of it.

Duskiness or lividity of the surface appears to increase, and it reaches its maximum just as the clonic spasms begin to abate in their severity, and the second stage passes into the third. The sweating is often excessive, and in some cases has been observed to have a peculiarly fœtid odour.

The pupils vary from contraction to dilatation, and back again, not, however, becoming so widely dilated as they were at the onset of the seizure; and they are, to some extent, influenced by exposure to light.

The veins are greatly distended, especially those of the throat; the heart beats tumultuously; friends of patients say, "It seems as if it would beat through the chest;" the carotids throb, and the arterial pulsation everywhere is violent, and the vessels are full.

In the *third* stage, there are many of the phenomena of the second, out of which it is gradually developed,—that which marks its arrival being the partial return of sensation, consciousness, and voluntary power.

The movements now witnessed are not wholly meaningless; the patient makes an attempt to change his position, or to do something, his efforts often, however, being frustrated by some violent spasm; he "looks" at those around him, with a bewildered, suspicious, or sad expression; still there is "expression;" and he may make some attempt to speak; the respiration becomes less unruly, he can clear his throat; the pupils are contracted, but he can see; the conjunctivæ are injected, and there are often petechiæ on the forehead, the temples, behind the ears, and in the eyelids; the pulse is variable; there is a jaded, exhausted state, and the patient seems tired and disposed to sleep.

This third stage may last from a few seconds to ten minutes, when the "after-stage" of stupor sets in. Often there is a confused mental condition, with occasional involuntary movements, lasting for several hours: often the patient recovers rapidly, and goes on with what he was doing before the attack occurred. There is, indeed, almost every degree of severity in the seizure; sometimes all the symptoms I have mentioned being passed through in a far shorter time than it takes to describe them; sometimes each stage being prolonged, and the patient passing gradually into a condition of stupor, from which he awakes, even after many hours, jaded and "beaten," and from which it takes several days for him to recover.

Vomiting often follows the attacks in many individuals; in some it is a constant sequence. Large quantities of pale urine are secreted in the majority of cases; both the urinary water and the amount of urea are increased; and deposits of uric acid and of urates may be discovered. I have, however, failed to find either sugar or albumen in the urine of those epileptics who were not affected by either diabetes or Bright's disease.

The *after-symptoms* of an epileptic paroxysm vary widely in character, severity, and duration. There is usually lassitude and stupor, with headache. It is difficult to rouse the patient, and, if awaked, he is often peevish and irritable, and sometimes suspicious. The sleep is usually tranquil, but occasionally disturbed, as if by dreams. There is commonly stertor, coming and going, guttural in tone, and unlike the noise made by mucus, rattling in the trachea, during the second stage. The muscular condition is that of relaxation, occasionally interrupted, for a moment, by clonic spasm or fibrillar contraction. This stupor may last, if the attack has occurred in the evening, throughout the night, passing insensibly into ordinary sleep. But when the seizure has taken place in the day-time, its average duration has been one hour. It has not appeared to me to bear any constant relation to the severity of the attack as measured by the violence of convulsion. It is often absent in lunatics who are subject to Epilepsy;[1] but Messrs. Leech and Fox show that there is some relation to be observed between the interparoxysmal and the post-paroxysmal mental state. When the post-paroxysmal symptoms are absent or slight, 38·7 per cent. are in the first mental class; whilst of those in whom these symptoms are slight, only 18·1 per cent. are free from interparoxysmal mental change.[1]

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[1] Dr. Bucknill, Asylum Journal, for October 1855.

M. Voisin states that epileptic fits produce changes in the sphygmographic tracings of the pulse, which last for several hours after the attacks, viz. ascending lines of great height and well-marked dicrotism.[2]

(*d*) *Epilepsia Abortiva, or Epilepsia Gravior without complete Loss of Consciousness.*—It is for the sake of practical convenience, rather than because it is strictly speaking pathologically correct, that the class of cases now to be described are mentioned in this place. Names, as employed in the science of medicine, are useful modes of recognition, and not exhaustive descriptions of the maladies they denote. We must give names to the diseases we describe; we must define what we denote by the names we use; yet, in so doing, we draw, besides the necessary, some artificial lines; and it is occasionally the least of many evils to overstep them.

The attacks to be described are almost excluded by our definition of Epilepsy, yet they so closely resemble that disease in all their own features, that they find a more fitting place in this portion of a System of Medicine than they could find elsewhere. They are closely related pathologically, and we find in their position here an example of the general principle of terminology employed in this work, and no departure from its spirit.

Abortive attacks of Epilepsy have been described by Dr. Prichard (Treatise on Diseases of the Nervous System, p. 91); M. Doussin Dubreuil (De l'Epilepsie en général, p. 16); Schr. van der Kolk (Pathology of the Medulla Oblongata, Syd. Soc. Transl., p. 211); Maisonneuve (Recherches et Observations sur l'Epilepsie, p. 22); Dr. Radcliffe (Epilepsy and other Convulsive Affections, p. 164); Herpin (Du Pronostic et du Traitement de l'Epilepsie, p. 429); Messrs. Leech and Fox (op. cit. p. 226): and M. Brown-Séquard has detailed the occurrence of similar phenomena in animals (Journal de Physiologie, tome i. p. 474). Several cases of seizures of an abortive character have fallen under my own observation; and what is to be said about them will occupy but little space. There has been sudden tonic spasm of the face, neck, and chest, accompanied by arrest of respiration, and followed by clonic convulsion, having the general form of an ordinary epileptic paroxysm; and yet there has been either no interference with consciousness, or only such slight obscuration as to be at first completely denied by the patient. Such paroxysms may occur, at intervals, for many years; they may take place in those who are subject to ordinary epileptic attacks; or they may exist in connexion with other signs of disease in the nervous centres.

The *interparoxysmal* symptoms of Epilepsy may be divided into those pertaining to the nervous system, and those not so related. The

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[1] Op. cit. p. 229.

[2] Ann. d'Hygiène, xxix. p. 358, quoted in Syd. Soc. Biennial Retrospect, 1867–8, p. 471.

"nervous" phenomena exist in regard of mind, sensation, and motility, and they are of varying intensity, prevalence, and kind. The most important are those which belong to the mental history, and they will be considered first.

(*a*) *Mental Condition of Epileptics.*—A prevalent belief is that some form or degree of mental deterioration is necessarily associated with Epilepsy. The result of inquiry upon this point is to show that there is no such "necessary" relation. The general belief is, however, to be accounted for partly by the strong impression which some notable cases of mental failure have made upon the minds of those who witnessed and recorded them,—such strong impression being followed by an undue inference,—and partly by the fact that the words Epilepsy and Epileptic have been made to include every form of disease of brain, spinal cord, or other organs, and also every variety of that multiform derangement which we call "insanity of mind" which might be associated with fits. It is desirable, again, to assert that this article refers only to such cases as constitute Epilepsy proper; and that the statistics upon which my results are based, can only with a double injustice be compared with those derivable from lunatic asylums. A patient may be epileptic and a lunatic; he may be epileptic and asthmatic; but there are some epileptics whose minds are as healthy as their lungs; and, so far as the natural history of Epilepsy generally is concerned, it is a mistake to derive it from complicated cases.

The mode in which I have endeavoured to answer the question,—what is the actual mental condition of epileptics during the intervals of their attacks?—has been the following. I have divided epileptics into four classes: in the first there are placed those in whom, neither by the patients themselves, by their friends, nor by myself, could there be detected any deviation from mental health; such individuals had "nothing the matter with them," but exhibited for their station in life and educational advantages the full average amount of intellectual vigour and cultivation. The second class consists of those who presented that slight defect of memory which is limited to the occurrence of recent and trifling events, the memory for those long since past being intact; and in those who formed this group, such impairment of memory was the only departure from mental health. In the third class are those cases which present, in addition to the loss of memory described, some diminution of the power of apprehension. Such patients are dull in acquiring new ideas, and often receive incorrect or imperfect and confused notions of what is brought before them. The fourth class includes those who, in addition to the failures exhibited by the preceding groups, are habitually confused, and unable to follow out any train of thought; people who seem to think little, but to be in a vacant, wandering state of mind, often idle, stupid, and indifferent, and sometimes almost or completely demented.

Having determined upon this principle of arrangement, it is comparatively easy to answer many questions of interest with regard to epileptics, and to state the answers to such questions in numerical terms. This I have done in another work;[1] and all that it is thought desirable to do now is to give some of these results,—and with them others based upon a wider range of facts,—without burdening the reader with a number of statistical details.

In rather more than one-third of all the cases which I have examined there has been perfect (*i.e.* average) mental integrity; in a little less than two-thirds, there has been some intellectual deterioration, but this has existed to a high degree in only a very small proportion. Women have been found to suffer more frequently and more severely than men; and the commonest form of failure is that of defective memory; this faculty being diminished, especially in regard to recent and trifling events.

It is of much interest to know the conditions which determine mental failure in the epileptic, and thus to avoid certain errors which are prevalent upon the subject. The results of inquiry upon this point may be stated in the following propositions:—

Hereditary taint is without influence.

The age at which Epilepsy commences exerts a certain amount of influence, and to this effect—that the disease when appearing late in life is more commonly associated with mental failure than it is under the opposite condition; and that the chances of mental failure are less when the attacks commence before the arrival at puberty than they are when Epilepsy is developed after that epoch. This statement is supported by the further statistics of Messrs. Leech and Fox.[2] Late rather than early Epilepsy is a predisponent to intellectual failure, and this whether we divide the cases at the tenth, sixteenth, or twentieth years, and whether we consider the two sexes together, or each sex separately.

The duration of Epilepsy is, *per se*, without influence upon the mental condition of the epileptic.

The amount of mental deterioration is not in direct proportion—but in inverse ratio—to that of muscular disturbance, as shown by the presence of tremor, or spasm, either clonic or tonic.

The state of the "general health" does not account for that of the mind; the former may be good, and the latter bad, and *vice versâ;* and, contrary to what would be expected, such relation is more usual than the co-existence of marked failure or integrity in both directions.

The number of attacks does not determine either the degree or the existence of intellectual change.

Frequency of recurrence of the seizures is, however, associated with mental change; but in such manner as to show that it is not the sole condition of such result, and that it is not even a necessary condition.

The severity of the convulsive paroxysm is without apparent influence, when such severity is judged of by the duration of subsequent coma. The form of the attack appears, however, to exert a considerable influence. Neither seizures of "le haut mal," nor those of "le petit mal," necessarily induce the change of which we are speaking; but the mental deterioration of epileptics is much more clearly associated with the minor than with the severer seizures.

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[1] Auct. op. cit.

[2] Op. cit. p. 212.

The nature of the exciting cause, viz. its existence in the psychical or the material elements of life, appears to be without influence in the determination of mental change.

(*b*) *Sensorial Condition of Epileptics.*—Headache and vertigo are the two forms of disturbance the most frequently complained of by epileptics. They exist, however, to a high degree in only a small number of the cases; and, when they do exist, have no special character which renders them of value in either diagnosis or prognosis. Headache is more frequent in females than males. The vertigo of epileptics is commonly of such kind that the patient rarely imagines that surrounding objects are in motion, but rather that he is, himself, moving or turning round; he feels as if he were doing so, and is unsteady in standing, or in his attempts to walk.

The pupils are more commonly beyond than below the average size; the special senses exhibit neither constant, prevalent, nor characteristic change.

(*c*) *Condition of the Motorial System in Epileptics.*—Some patients exhibit a tremulous state of the muscles; some, either with or without tremor, are affected by clonic spasm; others present tonic spasm, or cramp; whereas many are quite free from either of these forms of altered motility. In the majority of cases there is some kind of disturbance; but in the greater number of this majority the amount of such disturbance is slight.

The patient often says that he is "nervous," meaning by this that his hand is unsteady, or that the body is tremulous, or that he feels as if they were so.

It is often denied that any jerking of the muscles ever occurs; but the physician may frequently discover that such denial is incorrect. The amount of clonic spasm may be, therefore, very slight: it may, on the other hand, be very considerable, assuming one or both of two general forms. There may be, more or less constant and considerable, choreiform movement; and this may be observed not only when the patient is awake, but when he is asleep, and often with exaggerated force in the latter condition. There may be violent spasmodic shakings of the limbs or of the trunk; occurring at irregular intervals, but exhibiting an especial frequency of occurrence just as the patient falls asleep. Such jactitations have proved excessively annoying in several cases, and have been so troublesome as to entail much ulterior distress from the loss of sleep that they have occasioned. Sometimes the jerk of muscles is so sudden and so violent that the patient is thrown out of bed; or, if standing, is thrown down.

Cramp, or tonic contraction, is comparatively rare, and has apparently only an accidental relation to the disease. Messrs. Leech and Fox found it more frequent in occurrence than I have done (op. cit. p. 216).

(*d*) *Condition of the General Health.*—There are no changes in the "general health" of epileptics to be observed with such sufficient frequency or speciality that they deserve to be reckoned among the characteristic features of the disease. Epilepsy may exist in every condition of the general health; but among those who have been primarily poor, or who have become so owing to their disease, a low state of vitality is encountered. A similarly depraved condition may be found where the circumstances have been different; but such state is by no means necessarily present. Epileptics may be found in robust as well as in feeble health; but it is important to know the relative frequency of the one and of the other condition.

Patients have been examined by myself in regard to their nutrition, temperature, and strength, and the general results of such inquiry are those stated above. But, further, cases have been divided into four groups, viz.: into, 1st, those exhibiting, in every particular, good health; the limbs being well nourished, of normal temperature, and of natural strength;—individuals capable of enduring both exposure and fatigue, as well as any others of their age, sex, and social condition; 2d, those in whom some failure in one of the above particulars was noted; 3d, those in whom a double deterioration was observed; and 4th, those in whom there was deficiency in all three particulars. The result of such inquiry has been to show that more than one-half of the cases belonged to the first group; less than one-third to the second; less than one-tenth to the third; and little more than one-hundredth to the fourth. The most frequent change has been defective temperature; the least frequent, impaired nutrition. The pulse has exhibited no constant feature, either in frequency, force, or fulness.

There is, according to my experience, an entire absence of any specific change in epileptics, so far as regards their functions of digestion, respiration, circulation, and secretion.

If, as the result of this mode of inquiry, we regard epileptics as a whole, and put together all the results that have been obtained, we come to this important conclusion, that in a certain number (12 per cent.) there is nothing, absolutely nothing, abnormal to be discovered during the intervals of attack; that in nearly two-thirds of the cases some failure may be observed either in mind, motility, or general health; and that in less than one-third there is marked alteration.

It is then obvious that Epilepsy is a disease characterised only by its paroxysmal symptoms, and having, in the present state of science, no special features by which it may be recognised during the intervals of attack.

4. Relations between the Symptoms of Epilepsy.—(*a*) *Forms of Attack.*—The severer seizure, Epilepsia gravior, is nearly twice as common as the milder, Epilepsia mitior; and the former is much more frequently found by itself than is the latter. Hereditary taint seems to exert an influence in predisposing to the severer form of attack.

The milder attacks, however, do not appear to take the place of the more severe, but to be found with especial frequency in those cases which exhibit a rapid recurrence of the latter, *i.e.* of Epilepsia gravior. The form of attack does not appear to be determined solely, or even notably, by the age at which the disease commences; but when Epilepsy is developed early in life, there is an increased proclivity to the attack in its milder form. Duration of the malady does not determine its form of seizure.

(*b*) *Frequency of Attacks.*—In about one-seventh of the cases that I have examined, the seizures have exhibited a mode of recurrence which has been termed "serial;" that is to say, that the patients suffer from two, three, or more attacks in one day, and then pass through a period of freedom lasting from one to several weeks; and this mode of recurrence is more frequent in the female than in the male sex. The series, groups, or, as they are often termed, "bouts" of the fits, usually occupy one day only, and they are often limited to a period of twelve hours.

It is rare, very rare, to find an accurate periodicity in Epilepsy; but it is exceedingly common to observe that the recurrence of attacks has some kind of relation to time, as marked by its natural division into days, and periods of seven days, or multiples of seven days. Thus a large number of epileptics have their seizures every day, every two weeks, three weeks, and four weeks; while only a much smaller number suffer at such irregular intervals as cannot be thus expressed. An almost identical number of patients state that they have attacks at each of the periods mentioned; not meaning by that to say that there was always either perfect periodicity, or recurrence "to the day," but that, as a rule, every fortnight, three weeks, month, or day, there had been an attack.

There are four times as many epileptics who suffer from their seizures more frequently than once a month than there are of those whose attacks recur at longer intervals. The return of attacks at monthly periods is rather more common in the male sex than in the female; and it is very rare to find the seizures limited to the time of the menstrual discharge. It is frequently noticed that they are more common during menstruation; but, on the other hand, many women whose attacks recur at monthly intervals, exhibit no marked proclivity to their recurrence while the catamenia are present. A high rate of frequency is more common among women than among men.

The number of attacks in a given time ranges between very wide limits,—from two to two thousand in a year; but half the cases are found to have a rate of recurrence ranging from one attack in fourteen to one in thirty days.

Great frequency of attack is not constantly associated with signs of motor disturbance, such as tremor, clonic spasm, and the like.

Again, a high rate of frequency is not determined by an enfeebled state of the bodily health; but, on the contrary, is observed in those whose general physical condition is up to the standard of health, whereas a low rate of frequency is found in those whose organic powers have undergone marked deterioration.

An early commencement of Epilepsy is commonly, but not necessarily, associated with a high rate of frequency in the attacks. As the disease continues it exhibits a tendency to increase in the frequency of its paroxysms; but duration is not the sole condition determining this result.

(*c*) *Morbid Motorial Phenomena* are not found exclusively in those who exhibit an impaired state of the general health, but the one kind of derangement—marked by tremor, or clonic spasm—is commonly found in combination with the other, viz. diminution of temperature, or nutrition, or strength.

The prolongation of Epilepsy is not necessarily associated with impairment of the physical condition; but a high degree of the latter is often found in conjunction with a protracted duration of the disease.

(*d*) *Consequences of Epilepsy.*—If Epilepsy were found to entail, of necessity, any definite changes in the health of its subject, in regard of either mind, motility, or general condition, we should expect to find that such changes bore a definite and direct relation to the time during which the disease had existed. On this point, however, the result of a careful examination leads to the conclusion that duration is, *per se*, without effect, and that the demonstrable "consequences" are *nil*.[1]

5. COMPLICATIONS OF EPILEPSY.—These may exist in any organ of the body, but they have no such definite character, except when they are presented by the nervous system, as to require any special comment in this place. The most important is—

Epileptic Mania.—This complication occurs in about one-tenth of the cases, if we reckon all those degrees of such disturbance as may warrant the application of such name. Having occurred once in a particular individual, it is likely to appear again, and this is especially the case when several attacks have followed in rapid succession.

The delirium is commonly but not universally furious and dangerous; it is sometimes ecstatic in form, sometimes dull and melancholic. It may appear in the form of preternatural gaiety before the attacks, or in the intervals of their recurrence; it may break out as violent excitement just as the patient is emerging from the second stage of the paroxysm. Sometimes the mania has preceded the convulsions, but this order of events is, comparatively speaking, rare. Epileptics occasionally have some premonition of their maniacal state;—an indescribable feeling which leads them to place themselves under restraint before the occurrence of the outbreak. More commonly, however, there is no such warning, and the physician fails to discover any special reason for the attack.

Meningitis in an acute, sthenic form, may follow epileptic paroxysms; but when it has done so, it has, in the majority of cases, been determined by some accidental injury inflicted by a blow or fall, which the patient has experienced in one of his attacks.

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[1] Auct. op. cit. p. 199 et seq.

Apoplexy is so rare a sequence of Epilepsy that it is mentioned simply for the purpose of stating this fact, because it—apoplexy—is one of the dangers often quite unnecessarily dreaded by both epileptics and their friends.

Idiotcy may be complicated by Epilepsy; but when the two conditions are found together, or are stated to co-exist, the truth appears to be this, that the idiotcy has been congenital, and that the idiot has been "subject to fits." Abundant facts and reasons have been already furnished for the purpose of proving that the mode of regarding Epilepsy proper which would show that idiotcy is one of its frequent complications is fallacious, inasmuch as it widens the meaning of the word Epilepsy beyond what is pathologically correct, or practically desirable.

Convulsions, such as those attending upon dentition or parturition, exhibit no special frequency of occurrence in epileptics.

Paralysis is so rare an event that it may be regarded as having—like meningitis—an accidental, rather than essential, relationship to the disease in question.

Cyanosis is often accompanied by fits, and these have often assumed an epileptic character; but cyanosis is a rare malady, and its mere mention as a complication is all that is necessary here.

PATHOLOGY.—Anatomical investigation has hitherto failed to give any explanation of Epilepsy; every kind of lesion has been discovered in every organ of the body; and, on the other hand, every organ and part of organ has been found in perfect health. The observations of Wenzel,[1] those of MM. Bouchet and Cazauvielh,[2] and the later researches of Dr. Schröder van der Kolk,[3] have shown the existence of disease in the pituitary body, in the white substance of the brain, and in the medulla oblongata; but the changes that each of these authors has described have been found to be inconstant, and some of them quite exceptional. We must, therefore, admit the disease to be what is termed "functional," using that word in the sense strictly defined in the first volume of this work.[4] It is believed that "nutrition" is changed, but that its alterations are too fine for detection by our present modes of examination.

Bearing in mind all the facts of Epilepsy, and proceeding to their interpretation by the aid of physiology, we arrive at the following conclusions:[5]—

1st. That the seat of primary derangement is the medulla oblongata, upper portion of the spinal cord, and vaso-motor system of nerves.

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[1] Observations sur le Cervelet, &c. traduit par M. Breton.

[2] Archives Générales de Médecine.

[3] On the Minute Structure and Functions of the Spinal Cord, Syd. Soc. Transl.

[4] See p. 3, et seq.

[5] The reader is referred to the Author's Treatise on Epilepsy, chapter Pathology, for a full exposition of the views here stated propositionally, and also for complete reference to the different authorities quoted in support of each proposition.

2d. That the derangement consists in an increased and perverted readiness of action in these organs; the result of such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally.

By contraction of the vessels, the brain is deprived of blood, and consciousness is arrested; the face is, or may be, deprived of blood, and there is pallor; by contraction of the muscles which have been mentioned, there is arrest of respiration, the chest walls are fixed, and the other phenomena of the first stage of the attack are brought about.

3d. That the arrest of breathing leads to the special convulsions of asphyxia, and that the amount of these is in direct proportion to the perfection and continuance of the asphyxia.

4th. That the subsequent phenomena are those of poisoned blood; *i.e.* of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen.

5th. That the primary nutrition-change which is the starting-point of Epilepsy may exist alone, and Epilepsy be an idiopathic disease, *i.e.* a *morbus per se.*

6th. That this change may be transmitted hereditarily.

7th. That it may be induced by conditions acting upon the nervous centres directly, such as mechanical injuries, overwork, insolation, emotional disturbances, excessive venery, &c.

8th. That the nutrition-change of Epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ, rheumatism, gout, syphilis, scrofula, and the like; and further, that it may often be associated with change in the cortical substance of the hemispheres of the brain.[1]

9th. That it may be induced by some unknown circumstances determining a relative excess of change in the medulla, during the general excess and perversion of organic change occurring at the periods of puberty, of pregnancy, and of dentition.

10th. That it may be due to diseased action extending from contiguous portions of the nervous centres or their appendages.

11th. That the so-called epileptic aura is a condition of sensation or of motion dependent upon some change in the central nervous system; and is, like the paroxysm, a peripheral expression of the disease, and not its cause.

DIAGNOSIS.—Bearing in mind all the features of this disease as they have been described and limited in the foregoing pages, it will be comparatively easy to distinguish Epilepsy from every other malady.

The disease may be *simulated,* and when such is the case the fraud may be detected by the "over-acting" of the pretender, and longer duration of the paroxysm; by the choice of locality for the purposes of display; by the absence of those changes in colour which have been described; and last, but most certainly, by the absence of dilatation of the pupil. After the attack the sphygmograph may be employed in the manner adopted by M. Voisin.

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[1] See Dr. Wilks's paper in Guy's Hospital Reports, 1866.

Syncopal Attacks often resemble closely those of "le petit mal;" and the latter may be mistaken for the former. There is, perhaps, a much closer analogy between them than is sometimes supposed. For practical purposes of prognosis and of treatment, the distinction will turn upon these points of difference; in Epilepsy loss of consciousness is sudden, absolute, and often without any sense of "faintness;" recovery is rapid, and there is no recollection of the attack.

Hysteria, when convulsive in form, differs in the presence of some volition, some sensation, some power of directing movements. The attack is "got up," or passed into, gradually; and is preceded by sobbing, crying, laughing, and gesticulations: it continues sometimes for an indefinite period, and passes off through a stage of hysterical excitement.[1] The history of the case before the attack, and after its occurrence, is that of hysteria; whereas in Epilepsy there is or may be nothing abnormal to be discovered. In the attack there is not the hideous distortion of the features, neither is there the meaningless eye, nor the dilated pupil, nor the bitten tongue; respiration may be and generally is disorderly, but there is no marked asphyxia. After the attacks the patient is exhausted, but does not pass into stupor; hysterical mania or paralysis may follow, but they have their own special features.

Convulsions—such as those of teething, of worms, and the like—differ as widely from Epilepsy as attacks of bronchial catarrh do from genuine spasmodic asthma. The presence of dyspnœa, cough, and expectoration does not constitute a case of asthma; the loss of consciousness and convulsion does not constitute a case of Epilepsy. The real nature of the disease must be determined by those facts of its history which lie behind these symptoms, and determine its position in nosology. Convulsions may occur many times, and may sometimes pass into the disease we are describing; but they do not necessarily do this, and mere periodicity of recurrence is not the only mark of distinction between them.

Convulsions are most frequently found during infancy, and especially so while the child is cutting its first set of teeth. It is rare for Epilepsy to date from so early a period. Usually febrile symptoms precede the attack, or there is some definite source of irritation in the mucous membrane or secreting organs—*e.g.* dentition, worms, indigestion, scybalæ, calculi. The first occurrence of the convulsion and its subsequent repetition may be traced to one or more of the irritations enumerated. The attacks cease on the removal of their "exciting cause;" and they differ from Epilepsy in the following features:—Their invasion is less sudden, and the paroxysm is of shorter duration; there is not absolute loss of consciousness at the onset of attack; if perception, volition, and sensibility are entirely removed, such removal is during the clonic spasm, and not at the beginning; there is little or no subsequent stupor, and no paralysis.

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[1] See article on Hysteria.

The diagnosis of *diathetic* convulsions is based upon a recognition of the diathesis. At the onset of some of the exanthemata convulsions may occur, and assume an epileptic form, but they are to be distinguished by the fact of their appearance in early life, the patient being usually under six years of age; by the presence of febrile disturbance, and of some exanthem, or some acute inflammatory change such as pneumonia or bronchitis.

In "Bright's disease" of the kidney convulsions of epileptoid type may be the first symptoms which bring the patient under the notice of the physician. There will, however, be but little difficulty in establishing the diagnosis. There are marked and peculiar pallor, puffiness of eyelids or of ankles, and albuminuria. The attacks are followed, or have been preceded, by drowsiness, listlessness, and a tendency to delirium; there are headache, vertigo, clonic spasm, alternating with marked rigidity of limb, great irritability of the muscles on percussion, and often a highly characteristic state of the mental functions. The latter has these features:—The patient lies in apparently profound coma, with some limbs relaxed, and others rigid or in clonic contraction, breathing heavily with a stertorous sound, which may be found to exist in the mouth, and not in the throat; but, from this state of apparently profound stupor, he may be readily aroused to do that which he is told to do, or to answer questions; and immediately afterwards he falls again into the state of stupor. His condition resembles somewhat that of a person poisoned with opium.

It is sufficient to mention such diseases as *chronic alcoholism, lead-poisoning, syphilis,* and *rickets,* in order to indicate the means by which, when they are attended by convulsions, the diagnosis may be established.

Organic Diseases of the Nervous Centres may be distinguished from Epilepsy by the fact of their presenting symptoms over and above those proper to the latter. When conspicuous and persistent changes in the functions of the nervous system occur during the interparoxysmal period, we may infer the existence of structural disease. Again, there is more marked impairment of the general health; and the signs of disordered nerve-function have a more rapid development than have those which may occasionally be observed in Epilepsy. *Tumour* of the brain exhibits its most characteristic feature in persistent, or paroxysmally exaggerated pain, limited to a particular locality, and accompanied by local paralyses. *Chronic softening* may be diagnosticated by the gradual failure of mind, sensibility, and muscular power. *Chronic meningitis* may have a protracted history, but it is one of highly marked interparoxysmal change. There is irritability of temper, and, occasionally, delirium with loss of memory and impaired intellectual power: there is spasm alternating with local paralysis; and there are alterations of the special senses, with headache and general malaise.

The convulsions which occur in chronic cerebral diseases are not precisely like those of Epilepsy; there is less suddenness in their invasion, there is not the complete loss of consciousness, the convulsive movements do not pass through the several stages that have been described, but are irregular in their manner of development, protracted in their duration, and often limited to one side, or to one extremity. There are not the asphyxial phenomena of Epilepsy, neither is there the subsequent stupor.

Again, the ages at which intra-cranial diseases are developed differ from the prevailing age at which Epilepsy makes its appearance; neither aneurism nor carcinoma appears, as a rule, so early in life as does the disease under consideration; cerebral tubercle, when occurring in childhood, has a history widely different from that of Epilepsy; and, lastly, each of these is attended by its own special dyscrasia, which may afford all that is needed to complete a diagnosis.

PROGNOSIS.—When the disease has been established for some time, and is recognised to be an idiopathic affection, the prognosis is unfortunately very unfavourable as regards perfect and permanent cure. When it is recent, much hope may be entertained. Cases of eccentric convulsions and of chronic meningitis, either syphilitic or simple, may be cured, and such are often spoken of as epileptic; but I do not include them in the present article: the remarks here made apply exclusively to Epilepsy proper.

The *general prognosis* is framed upon several different considerations. Hereditary taint is of unfavourable omen; whereas an early commencement of the disease is the reverse. The duration of the malady is of the highest importance; the longer that it has lasted the greater is the difficulty and improbability of cure. Those cases in which the intervals between the attacks are much prolonged are less amenable to treatment than are those which exhibit a more rapid recurrence. Mental failure is of evil augury, but not to so high a degree as has been supposed. Some of the most obstinate cases are those in which the general health is good; some of the most tractable are those in which there is a disturbance which may be corrected.

Next in importance to that of the prognosis of the disease as a whole, is the forecasting of *the mental state*, supposing that the disease itself cannot be cured. What conditions are there which would render mental failure probable? The section on "natural history" supplies the answer to this query, but its results may be recapitulated here. Hereditary taint is without influence; the female sex is of unfavourable omen; late commencement of the disease is a predisponent to intellectual failure; mere duration is without influence; an impaired state of the general health is of good rather than evil import: mere number of attacks is of no moment; rapid recurrence of seizures is indicative of danger; and attacks of "le petit mal" are more injurious than are the severer paroxysms.

The *danger to life* is somewhat remote, and need scarcely be entertained. It is excessively rare for an epileptic to be killed by, or die in one of his attacks. Van der Kolk[1] has shown that the danger to life is greater in those cases in which the tongue is not bitten; but I have no observations to prove either the correctness or the incorrectness of this opinion, as I have never yet known a case in which the attack proved fatal.

TREATMENT.—There are two distinct elements to be regarded in the therapeutics of Epilepsy: the one is the diminution or removal of the condition which is the essential element in the disease; and the other is the mitigation of the paroxysmal symptoms when their removal cannot be effected. We have to direct the treatment of the disease and that of the attack.

It has been already stated that many epileptics, during the intervals of their paroxysms, present no abnormal condition; yet it is to be inferred that there must exist in them some departure from health, and the conclusion to which we have arrived is, that this departure consists in an undue readiness of action in certain portions of the nervous centres. Our object, therefore, is to control this over-readiness of action. For this purpose *sedatives* have been employed, and with success. It would be useless to attempt any estimate of the relative value of many of these agents, for there are no data sufficient for the purpose. Opium or morphia, conium, hyoscyamus, stramonium, belladonna, cannabis Indica, atropine, valerianate of atropine, selinum palustre, cotyledon umbilicus, chloroform, and other medicines have been employed with good effect in some cases, and without any appreciable effect in others, and hitherto no principle has been evolved from either their failure or success. When the attacks have been of very frequent recurrence, I have found preparations of the solanaceæ useful in diminishing the number of seizures, but I have never known them to effect a cure. Opium, or some preparation of morphia, has been of service when the patient was restless at night, and was obviously suffering from the effects of loss of rest. Chloroform has delayed attacks while the patient was actually under its influence, but has failed to prevent their subsequent recurrence. Dr. Murray has, however, been fortunate in the treatment of some cases by means of chloroform, and his observations are such as to warrant a further employment of this agent.[2] Indian hemp has relieved headache and restlessness, but has not cured or notably relieved Epilepsy.

The salts of zinc, and especially the oxide of zinc, have appeared to be of service in many cases; their action being obviously sedative. I have seen no good results from sulphate of zinc given in heroic doses, and the good effects that have come under my own observation have been from oxide of zinc in doses of three or five grains given three times daily. The salts of copper and of silver have proved utterly useless in my own experience.

Bromide of potassium, or some other salt containing bromine, is the one medicine which has, so far as I know, proved of real service in the treatment of Epilepsy. Undoubtedly it is "sedative" in its action; it lessens spasmodic movements, especially those of paroxysmal character, and sometimes ensures sleep when vegetable sedatives, and among them opium, have failed. Bromide of potassium in small doses has appeared to be of little or no service, but in large doses it rarely fails to give some relief. Sir Charles Locock has the merit of introducing this drug to the notice of the profession in this country,[1] and the testimony of all those who have had much experience in the matter concurs to a remarkable degree as to its utility. Given in doses ranging from ten to thirty grains, three times daily, it has had these effects:—In some cases it has completely cured the patient, and the cure has been permanent for years, and is so now. In others it has arrested the attacks so that none have occurred for periods varying from a few months to two or three years; but, on the omission of the medicine, the seizures have returned. In such cases the attacks have again ceased on the re-administration of the medicine. In a third series of cases it has diminished the frequency and severity of the seizures, but has not removed them altogether; the patients while taking the bromide have had one-half or one-third of the number to which they were habituated. Such patients have gone back to the old frequency of recurrence when the drug has been omitted, and have again improved when it has been re-administered. In a fourth, but very much smaller number, the influence of the drug has been good for a time, and has then appeared to cease; and in a fifth, and yet smaller proportion, it has been apparently without any appreciable effect. Still further, there are a very few cases in which the number of seizures has been increased by bromides. Dr. Duckworth Williams has shown that it exerts much influence over those cases in which the attacks take place during the day, but that it is of little use in those patients whose seizures occur during the night.[2] I have found that this is true to a certain extent, but not to the degree described by Dr. Williams; for in several instances KBr. has been very useful when the fits were limited to the hours of sleep. It is possible that Dr. Williams's cases may have been, from the fact of their complication with insanity, peculiar in this respect. Bromides appear less useful in growing girls and youths than in those who have reached adult age.

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[1] Op. cit. p. 252.

[2] Medical Times and Gazette, April 8, 1865.

It often happens that the administration of five grains will diminish the frequency of attacks, or prevent their occurrence, for a period of weeks or months; but that then, the medicine being still taken, the seizures revert to their previous rate of frequency. An increase of the dose is followed by a similar succession of events; a further increase by a second succession of temporary improvement and subsequent deterioration; and so on, until a larger dose, of from thirty to forty grains, is administered three times daily, when the attacks cease altogether.

It is not the mere administration of the drug, but its presence in certain quantity, that is necessary for a cure; but the dose which shall prove curative is not determined by either one of the following conditions:—sex, age, duration of disease, frequency of attack, severity of attack, or form of attack.

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[1] Lancet, May 20, 1857.

[2] Op. cit.

The number of cases in which it proves of no service, at any dose, is very small; and some of the cases which resist its action do not differ in any other obvious respect from those in which the bromide is highly efficacious.

In an earlier part of this article I have given reasons for thinking that the mode in which KBr. proves useful in Epilepsy is not by its diminishing either the sexual propensity, or power. It is positively curative of Epilepsy when given in doses which exert no influence whatever upon the generative functions.

In some individuals the administration of KBr. produces discomforts to which the term bromism has been applied. The most common of these is an acne-like eruption on the face, shoulders, and body generally; the most important is a state of stupidity and partial aphasia. Drowsiness, dulness of apprehension, muscular weakness, and general lethargy are often met with; and these symptoms may exist with varying degrees of severity, and be produced very easily in certain individuals. On the other hand, KBr. may be given for many consecutive years, and in large doses, without producing any one of the discomforts that have been mentioned. It is easy to remove these symptoms by a discontinuance of the drug, and the temporary administration of a bitter infusion with a mineral acid; and it is equally easy, and much more desirable, to prevent their occurrence, by omitting the medicine for one or two or even three days in the week. All the good effects of Br. are thus secured, and its evils are avoided.

Dr. Williams[1] states some facts which would appear to prove that KBr. does diminish the force of the heart's action; but in my own experience this has not occurred to any such degree, or with such frequency, as to make me attach any importance to its occurrence. Bromide of potassium has arrested Epilepsy without producing any diminution of cardiac action; and in all cases where there has been the least suspicion of such effect, the addition of chloric ether, or of tincture of cinchona, or indeed of any diffusible stimulant, has at once removed the threatened inconvenience. The bromide of sodium was suggested to me, some time ago, by my friend Dr. Ransom, of Nottingham, and I have now employed it in a large number of cases with perfectly satisfactory results. The dose is the same as that of KBr., but the NaBr. has this advantage, that it may be taken alone, as common salt, with food, and when mixed with an equal proportion of NaCl. would be quite unnoticed in the salt-cellar.

Counter-irritation, and derivants, such as setons, issues, and the like, have appeared to me to be of signally little service in genuine Epilepsy, so that I have been led to the belief that those cases in which they have been said to be of great utility have been examples either of some other malady, or of some complication of the disease.

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[1] Op. cit.

As to diet and regimen, these things seem to me important: first that the patient should eat digestible meals, with great regularity; and second that exercise, in the open air, should be taken as much as possible, short of fatigue. Many epileptics have been relieved from nocturnal attacks by being made to sleep with the head and shoulders well raised, not by pillows, but by a simple contrivance which is placed under the upper half of the bed or mattress on which they lay. Baths used for the purpose of cleanliness are useful in Epilepsy as in many other diseases; but I have seen more harm than good follow the employment of douche, shower, and sitz baths, when these have been administered in any manner or to any degree which exceeds that of producing comfort to the individual. Warmth to the extremities, especially at night, is of great value; the patient should never go to bed with cold feet, nor run the risk of their becoming cold during the night. Fires, hot water, hot-water baths, and woollen socks, may prevent much mischief. Sexual intercourse appears to me also to be one of those matters upon which the dictates of common sense are sufficient without any special direction from the physician.

The mental state of the epileptic may be much injured by action upon one very common form of advice, viz. that the patient should "do nothing." It is desirable to avoid over-exertion, worry, and undue excitement; but moderate mental exercise is of great utility; and some definite employment, carried to a point short of fatigue, should be enjoined as part of the treatment of those cases which are not complicated with cerebral excitement.

It is impossible to pay too great an amount of attention to the "general health" of epileptics, but there is nothing *special* in regard to this matter. Cod-liver oil, quinine, iron, alteratives, and aperients must be given in circumstances which would render their exhibition desirable in other forms of disease.

Allusion has already been made to the operation of clitoridectomy, and reasons have been given for suspending judgment on the matter. Doubtless success has followed such treatment in some cases, but the results are, at present, too uncertain for the formation of a definite opinion, first as to the stability of the cure; secondly as to the class of case in which the operation is justifiable; and thirdly as to the therapeutic *modus operandi* of clitoridectomy when it has appeared to be useful. It is not only possible, but highly probable, that an operation of severity equal to that of clitoridectomy might prove serviceable in some cases of Epilepsy if it were performed on the back of the neck, the mouth, or the toes. A strong impression upon the mind, or a violent change in the body, such as the opening of an issue, the performance of tracheotomy, or the occurrence of an accidental burn, has often arrested the attacks. It is probable that clitoridectomy and circumcision may, in some cases, act benefically in a similar manner; but it is obvious that, if they do, the form and locality of operation might be changed with advantage. So far as my own observation extends, the cases are almost infinitely rare in which such an operation would appear to me to be allowable.

The application of ice to the spine has, of late, been advocated with great ability by Dr. John Chapman; and there appear to be many theoretical considerations warranting the employment of this mode of treatment. It is not my purpose to detail the theory upon which Dr. Chapman has acted, as it is fully explained in his own writings. I regret to say that such application has utterly failed to do any good in a very large number of epileptics for whom I have prescribed it. Ice has been applied in the manner recommended by Dr. Chapman, and has been persevered in for many months, without producing the smallest effect upon the frequency or severity of the paroxysms. In one case, at University College Hospital, it was applied both night and morning, without influencing the disease, and on more than one occasion the fits took place while the ice-bag was on the spine. In one instance it was followed by relief, but in this case the patient was taking at the same time bromide of potassium. For the purpose of testing its utility I have employed it in a number of cases without giving any medicine whatever, and the result has been absolutely negative; it has done no harm, but it has done no good. It has appeared in several individuals to be of service in the first instance, out soon, in spite of its persevering application, the attacks have recurred with their usual frequency and severity. Patients have not complained of its application, but I have failed to find that it exerted any influence upon their temperature, when this was tested by their own sensations, or by the thermometer applied to either the axillæ or the extremities.

The *treatment of the attack* is mainly of value when directed towards its prevention; and there are several means by which some good may be accomplished. When an "aura" is present, the paroxysms may sometimes be arrested by cauterising the surface from which the aura comes, or by applying pressure between the starting-point of the aura and the trunk. Sometimes the attack begins by a special form of contraction in particular muscles, and its progress may be arrested by forcible extension of these muscles. Chloroform, or ammonia, if inhaled, will often prevent the seizures just at the moment of their onset; and in like manner a draught of wine, of sal-volatile and water, or of some other diffusible stimulant, will put off the attack. When patients have warning sensations, of sufficient duration for them to do anything, it is desirable that they should carry with them some little draught of this kind, which they may take at the moment of threatening. By such means a large number of fits may be averted.

Tracheotomy has been shown to be of no such real service in Epilepsy as to warrant its recommendation.

When the attack is once established, there is little that can be done beyond that of preventing the patient from injuring himself. Compression of the carotids may arrest or shorten the attack, but it does not cure the disease. It is possible that the pressure upon the nerve trunks is an important element in this mode of treatment. A piece of india-rubber may save the tongue from being bitten ; a loose cravat may diminish the petechial discoloration of the face ; and a strong arm may hinder the bruising of the extremities.

When the paroxysm is over the patient should be allowed to sleep, and should be placed with the head and shoulders raised.

In some epileptics the mental symptoms are the most highly marked features of the interparoxysmal period, and to these attention must be mainly directed. In others the general health is greatly at fault, and in them the treatment must be turned towards its improvement. In a third class there is excessive motility of involuntary kind, and in such cases the vegetable sedatives are of marked utility ; but in all, the medicine which has proved most useful, in my own experience, is the bromide of potassium.

MUSCULAR ANÆSTHESIA.

By J. Russell Reynolds, M.D. F.R.S.

Definition.—A loss of the feeling of muscular action, attended by irregularity, sluggishness, and diminished force of voluntary movement; but unattended by any necessary loss of cutaneous sensibility or by distinct paralysis.

Nomenclature.—The property which is diminished or lost, in the affection above defined, has been described under different names, of which the following are the more important:—"the muscular sense;" "le sentiment d'activité musculaire;" "le sens d'activité musculaire;" "la conscience musculaire;" "le sens musculaire;" "le sens de la force;" "le sentiment du mouvement;" "der Muskelsinn."

Symptoms.—The essential features of this condition are the following:—awkwardness and clumsiness in performing certain voluntary movements, sometimes of the hand and arm, sometimes of the leg, sometimes of the face. The patient tries to do what he wishes, or is told to do, and succeeds in the attempt by looking carefully at his limb, and helping it with one of the others which is unaffected; but if not paying great attention, or making any great effort, he fails to effect the movement, lets objects fall out of his hands, knocks his legs the one against the other, or in some other manner exhibits clumsiness and want of co-ordinating power. If placed in absolute darkness, or if the eyes are bandaged, he may be unable to execute any movement. The negative features are, that there may be no loss of cutaneous sensibility; the special senses may be intact; and there is no distinct paralysis.

Movements instituted in the affected parts are less vigorous than is natural; the limbs are somewhat inert, and often hang idly by the side or are carried by some mechanical contrivance; but they can be, by a strong effort, rendered almost as vigorous as in health, and the individual, after two or three awkward failures, may succeed in performing some complex act, provided that he thinks much about it and looks fixedly at what he is attempting to do.

If the muscles are pinched forcibly between the fingers, or if they are submitted to the electric current, they exhibit a diminution of sensibility. This has been well shown in a case lately under my care in University College Hospital; the patient did not know when the magneto-electric current was applied to the muscles of the right leg, although they could be seen to act quite vigorously. There was loss of cutaneous sensibility and of muscular power in the same limb, but electric irritability, although diminished, was preserved. As the voluntary power and the sensibility of the skin returned, some electric sensibility returned also, but it was notably deficient long after the electric contractility was almost normal; the patient being scarcely conscious of an amount of actual contraction of the muscles induced by electricity, which amount could not be tolerated for a moment in the muscles of the unaffected limb.

Without looking to see, the patient does not know the position of his limbs; and even when he has voluntarily assumed any attitude or position, he swerves from it if his attention be directed to some other object than his own limbs.

Minor degrees of this disturbance may often be observed in conjunction with definite but partial paralysis, in either a paraplegic or hemiplegic form. Such patients can only move their toes or fingers when they are looking at them; and they do not know, if their eyes are closed, whether they are moving their extremities or not, but, in perfect innocence, may ask the physician to inform them.

Commonly, Muscular Anæsthesia is seen in combination with other evidences of profound change in the nervous centres; but sometimes it exists, and that for a considerable time, alone. It may be, and often is, the precursor of paraplegia, and under such circumstances may be confounded with ataxy, spinal congestion, commencing myelitis, or softening of the cord.

The following case affords a fair illustration of the malady:—

A. B., female, at the age of 18 or 19 years, "caught cold" during menstruation, and soon afterwards felt "loss of power" in the legs and hands; she stumbled in walking, and found it very difficult to dress herself. The symptoms became slowly better, but occasionally returned; and three years after their commencement she married, and at the time of my seeing her, eighteen months after marriage, had a baby three months old.

She walked into my room leaning upon the arm of a friend, but she stumbled, and nearly fell down in doing so; her position in standing, when without support from another person or a table, was that of inclination forwards, and she rocked about from side to side, and antero-posteriorly: when she attempted to walk she occasionally reeled, and did this especially when engaged in conversation. If told to make an effort to walk in a straight line, she looked carefully at her feet and managed to do so without much deviation. When standing with her heels together she maintained steadiness of position so long as her hand was on the table, or she was paying attention to her drill; but, in a moment, if her mind was distracted by conversation, she staggered, and caught at some object for support. She told me that her hands were much better than they had been previously; but that still they were "very odd." It was, as I observed, difficult, and indeed impossible, for her to do sundry little things, such as putting a pin into her dress or taking it out, fastening or unfastening a button, without seeing either her fingers or the reflection of them in the glass. She found it impossible, or very difficult, to play on the pianoforte, and, as she expressed it, she "could not fasten anything she could not see." Objects fell out of her hands when she did not look at them; when standing with support on both sides, but with the eyes closed, she could not raise either foot from the ground,—the sole of the foot seemed glued to the carpet. The cutaneous sensibility was perfect; the electric contractility and sensibility were natural; there was no failure of general health, no tenderness of spine, no alteration in the special senses, no pain; and there were no symptoms of hysteria. All that was lost in this case was the sense of muscular condition and action.

CAUSES.—Nothing definite is known with regard to these, beyond the frequent association of Anæsthesia Muscularis with hysteria.

I once saw a marked case of Muscular and Cutaneous Anæsthesia which had been induced by exposure to cold. The symptoms in this instance were developed suddenly; but in other cases their commencement has been insidious, and their progress slow; and it has been impossible to assign any rational cause for their production. In many they have followed a series of convulsions or other symptoms of hysterical character.

DIAGNOSIS.—From *paraplegia* generally, whatever may be its cause, Muscular Anæsthesia may be distinguished by the facts that power is not lost, and that forcible movements may be determined, although not directed with exactness. In ordinary paraplegia the awkwardness of movement is due to and proportioned to the want of power; in Muscular Anæsthesia there is no such relation. Moreover, the patient exhibits none of the signs of interference with those functions of the spinal centre which are speedily involved in all cases of paraplegia depending upon changes in the nutrition of the cord. The limbs do not waste, the skin undergoes no special alteration, the urine is not altered, and sensation in other directions is unchanged.

From *hemiplegia*, indicative of those cerebral diseases which are usually accompanied by paralysis of one side of the body, Muscular Anæsthesia is separated by considerations similar to some of those which have just been mentioned; but mainly by the absence of conformity of the case to the known types of cerebral lesion, by freedom from intellectual change, and by the limited distribution of the symptoms.

Locomotor ataxy resembles Muscular Anæsthesia in its most striking symptom, viz. want of co-ordinating power; and in many cases of the former there are symptoms of the latter. It was present, for example, in 28 of 50 cases analysed by Topinard;[1] but it was absent in 22 cases, and was but slightly marked in 8. The clinical history of locomotor ataxy is different.[2] There is not necessarily the special want which is the essential condition of Muscular Anæsthesia; and in the latter there is an absence of pain in the limbs, of implication of the genital organs, and of affections of the eyesight.

Hysterical patients often exhibit the phenomena of Muscular Anæsthesia; indeed it is one of the expressions of their malady; and the only point of interest to ascertain is the degree to which this condition, the hysteric, may account for all the symptoms. The general course of the case usually affords the information that is required; it would be unsafe to refer Anæsthesia Muscularis to hysteria, unless other symptoms of the latter disease were present; it would be unwise to suspect the existence of grave central lesion unless hysteria could be excluded.

PATHOLOGY.—The present state of physiology with regard to the existence and nature of the muscular sense is so unsatisfactory that it would be quite idle to occupy much space in the discussion of its pathology.

With regard to the existence of such sense there appears to be evidence similar to that which we possess in respect of other senses, viz. our consciousness of its existence. It is a matter of fact that we do know when, in what direction, and to what degree we contract our muscles. We guide our movements without looking at our limbs, we know where our extremities are placed, we determine movements when we like, and apportion the amount of effort to the task set before us; we guess at the weight of a body by the effort we make to raise it, and do not break an empty egg-shell if we hold it between our fingers in the dark. The patient with Muscular Anæsthesia has lost the power or faculty which renders these adjustments of movement possible. The fact of the existence of a muscular sense may be regarded as established, and also that of its distinctness from all other modes of sensation. It is tolerably certain that the peripheral expansion of the muscular-sense nerves exists in the muscular tissue itself, and not in either the skin or the structures around the joints; but beyond this point there is grave doubt even as to whether the fibres pass in the anterior or posterior roots of the spinal nerves. M. Trousseau[3] admits the existence of muscular sensibility, but denies that of the sense of muscular activity; and the most important fact upon which he bases his opinion is contained in the following words: "Lorsque, fermant les yeux, nous exécutons sans efforts un mouvement assez étendu, il nous est impossible, avec la plus sévère attention, de sentir nos muscles se contracter; mais nous sentons le mouvement imprimé aux leviers que la contraction des muscles met en jeu. Le fait est si vrai, que si nous interrogeons une personne fort intelligente, mais complètement étrangère aux notions anatomiques et physiologiques, et si nous lui demandons quel est le siége du mouvement d'extension et de flexion des doigts, elle le place exclusivement dans la main et jamais dans l'avant-bras." This observation is quite correct, but M. Trousseau's conclusion from it is, I think, erroneous. We do not see objects, nor hear sounds *in* either our eyes or ears; but involuntarily project these sensations, not into a distant part of our own body, but into space outside ourselves. The senses of taste, smell, and of tact, we refer to something or somewhere just beyond the extreme peripheral expansion of the nerves which minister to those senses. We do not feel—or mentally recognise as such—the condition of our own nerves, but instinctively and of necessity feel and believe in something outside ourselves, or objective, that presses on the skin; something not ourselves that we taste in our mouths; something not ourselves that we smell in our noses. It is well known that a patient who has lost his leg imagines that he feels pain in his amputated toes, and in this we have another illustration of the principle that the mind does not refer sensations to the spot which receives the impression which may occasion it. Because, therefore, in the act of muscular movement our consciousness refers the sense of such movement to the extremity moved, and not to the moving organ, it is not proved that there is no sense of muscular activity; on the contrary, it is shown by this fact that the muscular sense obeys a law similar to that which we recognise in regard of other senses. For the existence of the sense we have the evidence of consciousness; and for the absence of the sense, there is the testimony of disease. It matters, comparatively speaking, little for our present purpose to determine the exact nature or metaphysical relations of the property in question; it is enough that in health there is a faculty which has been called "muscular sense," and that in disease this function is destroyed; that such disease may exist alone, and that the name by which it is denoted is "Muscular Anæsthesia."

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[1] De l'Ataxie locomotrice, p. 203; Paris, 1864.

[2] See article on Locomotor Ataxy.

[3] Article "Ataxie locomotrice progressive," Nouveau Dictionnaire de Médecine et de Chirurgie pratiques, tome 3me, p. 777.

PROGNOSIS.—The future of such cases cannot be predicted with certainty. It may be guessed at by regard to conditions other than those of the affection itself. If it be but one of many symptoms of that manifold disease called hysteria, the prognosis is that of the latter malady; if it be associated with grave changes in other portions or functions of the nervous system, the nature of such ulterior symptoms must determine the prognosis. There is nothing special in the character of the symptoms of Muscular Anæsthesia, *per se*, which can form a satisfactory guide.

TREATMENT.—Faradisation of the affected muscles has proved of service, as also has friction of the skin, and its electric irritation; but there are no medicines that have been shown to exert any special influence upon this variety of nervous disorder.

WASTING PALSY.

BY WILLIAM ROBERTS, M.D. F.R.C.P.

DEFINITION.—A chronic disease, consisting in a progressive atrophy of the voluntary muscles, independent of any antecedent motor or sensory paralysis. The disease attacks the muscles in groups: in some cases it is *partial*, and limited to the extremities; in other cases it is *general*, and implicates the muscles of the head, neck, and trunk.

SYNONYMS.—Paralysis Atrophica; Progressive Muscular Atrophy; Cruveilhier's Atrophy; Atrophie Musculaire Progressive (Fr.); Progressive Muskelatrophie, Progressive Muskel-lähmung (Ger.).

HISTORY.—Cases of extreme wasting of the muscles of the upper and lower limbs, without loss of voluntary power, were published in this country, in the earlier decades of the present century, by Cooke, Bell, and Darwall; but the establishment of the affection as a distinct type of disease is due to the labours of Cruveilhier, Aran, and Duchenne, in France, in the years 1851–53. The present writer collected all the information existing on the subject up to 1858, in an Essay published in that year.[1] To this Essay the reader is referred for the earlier notices of the disease. Since 1858 the pathology of Wasting Palsy has been elucidated by the investigations of Gull, Lockhart Clarke, Luys, and others.

ETIOLOGY.—The subjects of Wasting Palsy are mostly found among young adults and middle-aged individuals; but children are not unfrequently attacked. The mean age of eighty-eight cases collected by me was thirty years—the youngest was only two years of age, and the eldest sixty-nine. The male sex is considerably more liable to the disease than the female (about six males to every one female). This disproportion probably depends, mainly, on the greater and more sustained muscular exertion which men's occupations demand; also on the greater exposure to cold and external violence of individuals of the male sex. Women of the working-class—washerwomen, domestic servants, sempstresses, &c.—are seemingly not much less liable to Wasting Palsy than men employed in kindred occupations; but females belonging to the easy classes enjoy a remarkable immunity from this disease. It is, however, somewhat difficult to explain why cases arising from hereditary influence should occur more frequently among males than females.

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[1] An Essay on Wasting Palsy, by William Roberts, M.D. London, 1858.

Partial or *local* muscular atrophy prevails mostly among handicraftsmen — mechanics, masons, smiths, miners, needlewomen, scriveners, labourers, and domestic servants. The subjects of *general* Wasting Palsy are found equally in every grade of life.

The influence of *consanguinity* in the production of this disease has been remarked in a number of instances. The present writer collected the history of ten families in which a tendency to Wasting Palsy prevailed. In four of these families the disease was confined to two brothers in each. Dr. Meryon's first described cases were four boys who had six healthy sisters. In another family mentioned by him all the boys—namely, two—were affected, while the two sisters were healthy. A sea-captain, whose history is related by Aran, had lost two maternal uncles and a sister by the same disease. In another instance, recorded by the same observer, the patient's two aunts had died from general muscular atrophy; and, in a family known to Oppenheimer, two uncles and a cousin were already deceased, while another cousin and two brothers still suffered from the same disease. Altogether these ten families included twenty-nine individuals affected with Wasting Palsy, and of these only four were females. Cases arising from hereditary influence present another well-marked feature —in nearly all of them the disease became generalized, and consequently tended to a fatal termination.

As a rule, the subjects of Wasting Palsy have been persons of good physical development: in several cases the patients are reported to have been men of remarkable muscular power and activity; in a few instances—nearly all of which were associated with a hereditary proclivity to the disease—a certain weakness existed from early youth.

The exciting causes of Wasting Palsy (excluding hereditary predisposition) may be ranged under three heads; namely, excessive muscular action, cold, and disease or violence affecting the spine. In a considerable number of cases, however (36 per cent.), no reasonable cause could be assigned for the breaking out of the disease.

Aran directs attention to the fact that the particular muscles which are necessarily in long-continued contraction in persons following certain mechanical trades (masons, milliners, shoemakers, smiths, &c.), are those which are first invaded and most deeply involved. In persons of this class the muscles of the shoulders, arms, and hands are first affected, and very frequently the atrophy is permanently limited to these parts. There are numerous exceptions, however, to this rule.

Cases arising from cold (wearing of damp apparel, immersion of the limbs in cold water, rapid cooling of the perspiring surface, exposure to inclement weather) are marked by a train of neuralgic or so-called rheumatic pains in the affected parts, either at the onset of the atrophy, and ceasing when this has fairly set in, or continuing throughout its progress, and imparting a special character to the symptoms. The invasion of the disease in this class of cases is often somewhat sudden, and accompanied by cramps and twitches of the muscles.

In cases traceable to cold, the wasting is more apt to extend to the muscles of the trunk than in cases due to overwork. Of twenty-five cases attributed to overwork, eighteen were partial and only seven general; whereas, of the sixteen cases charged to the agency of cold, six were local and ten general. These two causes are often in operation together: the miners in my neighbourhood, who work in damp or wet excavations, are frequent victims of Wasting Palsy.

The connexion of Wasting Palsy with injury or disease directly or indirectly implicating the spinal cord, has of late years attracted increasing attention; and the interpretation of these cases has an important bearing on the pathology of the disease, as will be more particularly noticed hereafter. The history of some antecedent violence occurs too frequently in the reports of cases of Wasting Palsy to allow of its being set aside as a merely fortuitous circumstance, though the precise connexion between the injury and the subsequent atrophy is often obscure. In a youth under my care at the Manchester Infirmary, who ultimately died from implication of the respiratory muscles, the first symptoms of atrophy in the ball of the right thumb occurred six months after the fall of a bale of cotton-cloth on the nape of the neck. The immediate effects of the injury were confined to slight stiffness of the neck, and occasional pains extending down the arms. Valentiner records a case in which the first failure of health followed a fall on the back from a height of eight or ten feet: yet the atrophy did not appear until six years after. Bergmann's patient[1] fell on his back from a horse, and lay for a while unconscious. From this time he suffered pain and stiffness in moving the head; afterwards, and very slowly, a weakness in the shoulders came on, which ended in complete atrophy of the muscles around the shoulder joints. In a remarkable case recently reported by Dr. Thudichum and Mr. Lockhart Clarke, a gentleman, æt. 54, suffered what he considered a slight injury. In jumping across a flower-bed for a wager, he came down heavily on his heels, and then fell backwards upon his head. He was stunned for a time, but gradually recovered, and, after some days' confinement to his bed, appeared to be quite well again. It was, however, soon perceived that a great change took place in his habits. Having been extremely fond of manly sports and exercises—rowing, cricketing, riding on horseback, dancing, and the like—he discontinued to take part in any of these, although he continued to go every autumn to the Scotch moors for the purpose of shooting grouse. Five years after the above-mentioned accident, while engaged in this last-named sport, he perceived that his right leg had lost a part of its usual strength. From this time gradual atrophy and loss of power in the muscles crept over the patient, until at length death took place from failure of the respiratory muscles. Wide-spread degeneration of the spinal cord was found after death. (Beale's Archives of Medicine, 1863.)

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[1] St. Petersburger Med. Zeitsch., p. 116. 1864.

In other cases, disease, manifestly primary, of the spinal cord is followed by complete atrophy of certain groups of muscles. In a case published a few years ago in the *London Medical Review* by the present writer a young man suffered from acute general paralysis of all the muscles of the extremities, and of most of those of the trunk. The intellect was not affected. Gradually, in the course of months, the patient recovered the power of the muscles; but after complete restoration of the remainder of the body, the intrinsic muscles of both the hands and feet passed into a state of total atrophy, and still continue in the same condition. In the so-called essential paralysis of infancy and childhood—which is evidently of spinal origin—certain limited groups of muscles not unfrequently pass into a state of permanent atrophy, while the remaining portions of the paralysed members recover their mobility.

Certain other exciting causes of Wasting Palsy are sometimes doubtfully mentioned—namely, constitutional syphilis, venereal excesses, onanism, and antecedent zymotic fevers.

SYMPTOMS.—The invasion of Wasting Palsy is always gradual, and the disease has usually been in progress some weeks or months before the patient discovers its existence. The first symptom perceived is a certain weakness in the affected member: the tailor finds he cannot hold his needle; the shoemaker cannot thrust his awl; the mason fails to wield his hammer; the gentleman experiences an awkwardness in handling his pen, in pulling out his pocket handkerchief, or in putting on his hat. Some such incident calls attention to the affected limb, which is then usually discovered to be more or less wasted and shrunken.

The disease begins, in the great majority of cases, in the upper extremities, either in the ball of the thumb and hand, or in the shoulder—much more commonly in the former than in the latter. Sometimes, however, it begins in the muscles of the neck, of the face, the tongue, in the thigh, the leg, or the foot. The extension or spread of the disease follows an erratic course. In the immense majority of cases the disease is permanently limited to one or a few groups of muscles in the upper or lower extremities; in other cases, and these are by far more formidable, the atrophy invades successively the voluntary muscles of the entire body, trunk and extremities. The only muscles which, as yet, have not been known to be attacked, are those of mastication, and those which move the eyeball. When the atrophy is confined to certain regions of the extremities, the life of the sufferer is not imperilled; but when the trunk is invaded, and the muscles of respiration participate in the disease, death by suffocation is the ultimate result.

The wasting and disappearance of the muscles produce notable changes in the configuration of the body. The natural rounded contour of the limbs is replaced by an unsightly flattening; the bones stand out in unaccustomed distinctness, giving to the member the appearance of a skeleton clothed in skin; but the skin itself, and the subcutaneous cellular tissue, undergo no change, and cannot be distinguished from the integuments of healthy parts. Certain distortions of the head, trunk, and extremities are also occasioned by the unequal wasting of opposed groups of muscles—those less atrophied overcoming the resistance of those more diseased. These changes of configuration are a marked feature of Wasting Palsy. The hand is frequently the seat of a very singular deformity—namely, the "claw-shaped" hand, or "main en griffe" of French writers. The palm is robbed of its muscular cushions; flat planes or hollows occupy the sites of the thenar and hypothenar eminences; the hollow of the hand is traversed by the visibly prominent diverging flexor tendons, which are stretched between the wrist and the bulging bases of the fingers; the proximal phalanges are bent backwards, away from the hollow of the hand, while the middle and distal ones, inclined in an opposite direction, are in a state of continued semi-flexion. The back of the hand is hollowed out in long furrows, corresponding to the interosseous spaces, and the first joints of the fingers are pulled backwards, giving the hand a broken-backed appearance.

Passing up the limb, the forearm is found flattened, or even hollowed, on its anterior and posterior aspects. When the shoulders are affected, the whole arm dangles powerlessly at the side; the roundness of the shoulder has given place to a flattening, and the head of the humerus, the acromion, and the coracoid processes are plainly discerned through the thin covering of skin. If the serratus magnus be destroyed, the angle of the scapula is tilted upwards and inwards, and stands prominently out from the trunk. Corresponding deformities are witnessed when the lower limbs are invaded: the foot is distorted by the unequal involvement of its extrinsic and intrinsic muscles, and contractions of the toes on the sole, deflections of the foot inwards, or of the heel upwards, are produced—interfering very seriously with the steadiness of progression.

But perhaps the most remarkable of all the anatomical changes are seen in the face, when the muscles of expression are destroyed. The face is veiled, as it were, by an impenetrable mask; no emotion changes its unvarying aspect—the expression is always solemn, stolid, and unmoved. The muscles of the eyeballs are, however, spared, and by their movements alone, in the later periods, the mind holds an imperfect communion with the external world. The oral and buccal muscles are usually invaded early, and the saliva dribbles over the lips. When the muscles of the neck are involved, the head falls forward—the chin resting on the sternum—or, laterally, the head falling over on the shoulder.

When the abdominal muscles are implicated, the lumbar curve is enormously exaggerated by the unopposed action of the erector spinæ, and the belly projects in front, while the chest is thrown back as a counterpoise. The invasion of the lingual muscles leads to a falter in the speech, and to imperfect comminution of food in the mouth. The involvement of the laryngeal muscles produces a change in the voice, which loses its register, and is finally reduced to a monotone. When the diaphragm and intercostals are reached, violent suffocative fits of coughing are occasioned; the play of the chest is at length so reduced that a slight additional difficulty to respiration proves fatal. Dissolution is usually brought about by a bronchitic seizure; the air-tubes are speedily clogged with mucus, which no efforts of the patient can dislodge, and rapid asphyxia closes the scene.

When the disease is partial in its extension, it is observed that certain parts of the body, and certain groups of muscles, are much more obnoxious to its inroads than others. The muscles of the trunk are less liable than those of the extremities, and those of the lower extremities are far less frequently affected than those of the upper. Of sixty-two cases of partial Wasting Palsy collected by me, the upper extremities were alone affected fifty-one times, the lower extremities alone five times, and the upper and lower together seven times. The right arm was much more frequently attacked than the left, and the hands oftener than the shoulders. As a general rule, it was found that when one limb was attacked, its fellow of the opposite side shared its fate; that when the disease was unilateral, the right side was more likely to be its seat than the left.

One of the most striking characteristics of Wasting Palsy is the capriciousness of its line of attack. Scarcely two instances are exactly alike in the combination of muscles implicated—hence an almost infinite variety of feature; yet there are certain more common combinations.

Among the most common cases are those in which the disease is confined to the hands, or to the hands and forearms. Not uncommon, likewise, are the cases in which the shoulder and upper arm of one or both sides are atrophied, while the forearms and hands remain healthy.

Coincidently with the loss of substance in the muscular masses, there is necessarily a corresponding loss of power. Certain less constant symptoms also sometimes make their appearance—namely, fibrillary tremors, cramps, twitches, and diminution of electric contractility in the muscles.

The loss of power corresponds, in the typical cases, very exactly to the grade of muscular atrophy, and gradually proceeds as the muscles diminish in bulk. In extreme cases absolute immobility of the limb, or part, is at length produced; more commonly the various movements are still capable of being performed, but with greatly diminished force. Not unfrequently, however, this correspondence is not exact; and the loss of power exceeds, more or less considerably, what is due to mere atrophy of the muscular fibres.

During the active stage of the disease the affected muscles sometimes exhibit curious vibratile tremors—fugitive wavy oscillations of the muscular fibres—which are visible under the skin, but do not produce any movement of the limb, nor are they sensible to the patient. When absent, they may occasionally be evoked by stripping the part or filliping the skin. These vibrations are sometimes the earliest symptom of a new advance of the disease into parts not yet affected. They disappear altogether when the atrophy has reached an extreme degree, or when its progress has been arrested.

In uncomplicated cases the muscles of the wasting members respond to the electric stimulus readily, and with a force corresponding to their bulk. As a rule, there is no alteration in the tactile sensibility of the affected limbs; but in rare cases there is a slight numbness of the skin, and not unfrequently the parts are highly sensitive to impressions of cold. In about half the cases there is more or less pain of a neuralgic character in the course of the nerves leading to the diseased muscles, or in the neighbourhood of the muscles themselves. In some cases pain of an agonizing character is a marked feature of the complaint.

The general health is usually quite unaffected, the intelligence is clear, and the functions of organic life are performed with their usual regularity, so long as the muscles of deglutition and respiration are spared.

Course and Duration.—The course of Wasting Palsy is essentially chronic, and its duration uncertain. After destroying a certain group of muscles it may be permanently arrested, or it may proceed step by step until nearly all the voluntary muscles are disabled. The atrophied muscles may be again restored by therapeutical means to their original bulk: this is unfortunately not a very common termination—more commonly the wasted parts are crippled for the remainder of life. When the disease is progressive, its advance is seldom continuous, but is rather marked by repeated pauses and recommencements. The pauses may extend over a few weeks or months, or even several years. In a case now under my care in the Manchester Infirmary, the disease has started afresh in great intensity, after complete arrest for five years. In twenty-eight cases in which I was able to ascertain the continuance of the active process, the mean duration was thirty-eight months. Of these, four ended in recovery, thirteen in permanent arrest, and eleven in death. The cases which ended in recovery had a mean duration of fourteen months, those ending in arrest a mean duration of twenty-seven months, and those ending in death averaged a duration of more than five years.

Cases which could be traced to the effects of over-exercise of the muscles, were nearly always found to terminate in permanent arrest after the destruction of one or more groups of muscles; whereas cases which appeared to have arisen from exposure to cold, or from hereditary predisposition, showed a more decided tendency to a progressive course and a fatal termination.

DIAGNOSIS.—The partial form is liable to be confounded with paralysis from injury to a motor nerve, lead palsy, and malarious palsy. In all these there is a marked atrophy of the muscles; and the affection may be confined to a narrow region, around which are healthy muscles, offering a strong contrast to the decayed ones.

Atrophy, resulting from injury to a nerve, is distinguished by the exact limitation of the wasting to the parts supplied by that nerve; also, if the nerve be a mixed one, there is, or was, an accompanying loss of sensation.

In lead palsy there is a comparatively sudden invasion: in a day or two—a week, or a fortnight, at most—the paralysis is at its height; whereas in Wasting Palsy the loss of power is excessively gradual. The precursory or concomitant phenomena, distinctive of lead poisoning, seldom or never altogether fail—namely, colic, blue line on the gums, tremblings, pallor, and other symptoms of saturnine cachexia. Duchenne states, that the electric contractility of the muscles is markedly diminished or altogether lost in lead palsy; whereas, in Wasting Palsy, the muscles respond to the electric stimulus in a degree proportionate to their bulk. It will also be remembered that in saturnine poisoning the atrophy is distinctly sequential to the paralysis.

From ordinary general paralysis of central origin, Wasting Palsy is distinguished by the *dissecting* character of its march. It attacks the muscles in separate groups—in detail, as it were—and does not diffuse its ravages uniformly over extensive regions or the entire body. It is very rare also that in general paralysis the wasting of the muscular masses bears any proportion to the loss of power.

Extreme muscular atrophy sometimes follows infantile paralysis, and the distribution of the disease may resemble that of Wasting Palsy, and produce ultimate results indistinguishable therefrom. The cases are, however, totally different in their history. Infantile paralysis has always a sudden invasion, and the wasting is subsequent to the loss of power.

MORBID ANATOMY.—The essential changes found in the bodies of persons who have died from Wasting Palsy, are confined to the muscles, the spinal cord, and the nerves.

The muscles of the affected regions are found wasted in various degrees. Some are only slightly atrophied, others more profoundly, while others again are reduced to pale, thin, membraneous strata, or are altogether destroyed, and can only be identified by comparing the origins and insertions of certain fibro-cellular bands, which are the vestigial representatives of the previously existing muscular masses. The colour of the wasted muscles is changed to a pale red or rose, sometimes with a buff or ochreous tinge, and not unfrequently streaks of adipose tissue run, in lines, between the fibres. Where there is much fatty change, the wasting, which is so conspicuous a characteristic of the disease, is less marked; sometimes even the muscles are almost undiminished in bulk, but are transformed into masses of fat. This peculiarity has been observed only in the lower extremities.

The difference in the degree of atrophy undergone by adjoining muscles, and sometimes even by different parts of the same muscle, is very remarkable. Scarcely any two muscles are affected in an equal degree. Side by side with a pale, almost filamentous remnant, may be found a muscle of full red colour and undiminished bulk. One or two fasciculi of an affected muscle may survive in vigour after the total destruction of the remainder.

The decayed muscles have been examined microscopically by Meryon, Galliet, Oppenheimer, Virchow, and others. Meryon describes the primitive fibres as completely destroyed, the sarcous elements being diffused, and, in many places, converted into oil-globules and granular matter; whilst the sarcolemma was broken down and destroyed. Galliet, who examined the muscles in one of Cruveilhier's cases, states that in those parts of the muscle which had retained a rosy hue, the primitive fibres had preserved their striæ tolerably distinct, and between the striæ were seen fine grey or brilliant molecules, resembling fat. In the completely decolorized parts—those which to the naked eye appeared of a straw tint—there could still be recognised long cylinders, representing the primitive fibres. The sarcolemma was preserved, but the contained substance had lost its striated character, and was replaced by a uniform granular mass, presenting numerous minute grey molecules mixed with fatty granules. In parts where the disease was still further advanced, the granular matter and its enveloping sarcolemma had entirely disappeared, and there remained only the fibro-cellular framework of the muscle, destitute of any true sarcous tissue.

The condition of the spinal cord and of the spinal nerves has been examined in some thirty-five cases, of which thirty-four have been tabulated by Bergmann.[1] The results of the investigations have not been by any means uniform. In sixteen cases the cord and the nerves were pronounced healthy, and in six of these the parts were examined microscopically. In six cases the cord itself was found healthy, but there was marked atrophy of the anterior roots of a certain number of spinal nerves. In one case both the spinal cord and nerves were healthy, but there existed disease of the medulla oblongata. In six cases the cord was found diseased when examined microscopically, though it appeared sound, or nearly so, to the naked eye. Lastly, in seven cases the cord appeared to the unaided senses palpably softened and disorganized.

Atrophy of the anterior roots was first noticed by Cruveilhier, and was supposed by him to supply the key to the pathology of this disease. He thus describes the condition of these structures in the body of the showman Lecompte, who died from general Wasting Palsy of five years' duration. "The anterior roots of the spinal nerves are remarkably small compared with the posterior, and this inferiority of size is particularly great in the cervical region. The proportion between the two roots had become greatly changed. According to my observations, in the normal state, the posterior roots compare with the anterior, in the cervical region, as three to one; in the dorsal region, as one and a half to one; and in the lumbar region, as two to one. But here the proportion was as ten to one in the cervical, five to one in the dorsal and lumbar regions. Further, by plunging the cord into dilute nitric acid, I was able to observe that a very large number of the anterior cervical rootlets had been completely reduced to their neurilemma, and appeared as grey filaments, which, searched with a strong lens, presented no trace of nervous tissue; while, on the other hand, the anterior roots in the dorsal and lumbar regions had only suffered atrophy by emaciation. I was unable to trace the grey nervous filaments, or those simply atrophied, beyond the point where the anterior root joins the posterior; but I was able to establish the existence of atrophy of the nerves as they were about to penetrate the muscles."[1] A similar atrophy of the anterior roots was found in ten other cases, either with or without discoverable disease of the corresponding regions of the cord. In the great majority of the cases, however, the anterior roots were not perceptibly atrophied, and this leads directly to the inference that such atrophy is not an *essential* feature of the morbid anatomy of Wasting Palsy.

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[1] St. Petersburger Medicinische Zeitschrift, Bd. vii., 1864.

The morbid anatomy of the spinal cord is confessedly a subject of great difficulty. Until recently only the coarser changes of consistence—softening or induration—were appreciated by pathologists; and even after the microscope had been brought in aid of the examination, it soon became apparent that very important changes in the structure of the cord might be overlooked, unless the observer possessed special skill and practice in this branch of inquiry. The positive results of Gull, Lockhart Clarke, and Luys, who may be regarded as experts in the examination of the spinal cord, throw considerable doubt on the trustworthiness of the negative results obtained by Meryon, Savory, Oppenheimer, Friedberg, and others, who failed to detect in the spinal cords of patients who had died from Wasting Palsy any appreciable changes of structure.

Luys describes as follows the microscopical changes in the *apparently* sound cord of a man, aged fifty-seven years, who died of pneumonia, and who had been the subject of advanced atrophy of the muscles of the left hand and forearm. There was also slight atrophy of the muscles of the right hand. The loss of power had corresponded accurately with the degree of wasting. Five of the anterior roots coming off from the cervical enlargement of the cord were atrophied. The microscopic examination of the cord showed increase of the capillary vessels in the grey substance at the level of the atrophied roots. The walls of the vessels were thickened and surrounded with a granular deposit, which extended into the grey substance. In the anterior grey cornua, at the point of exit of the anterior roots, there was an absence of nerve-cells, which were replaced by granular deposit. Some of the nerve-cells of the anterior horns were in process of degeneration—brownish, and filled with dark granulations. These changes were found especially on the left side, and very slightly on the right side. The rest of the cord was healthy.[1]

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[1] Archives Générales, 1853.

Dr. Gull gives an account of a man, aged forty-nine years, who became the subject of Wasting Palsy after striking his head against a beam, whilst driving under an archway. Some months after this accident he began to suffer pain from the occiput down over the shoulders, and in about a year the muscles of the upper extremities began to waste. Three years after the accident he was admitted into Guy's Hospital. He then presented a remarkable example of muscular atrophy, without actual paralysis. The upper extremities were principally affected. The extensors of the right hand, the muscles of the thumb, and the interossei were extremely wasted. The wrist dropped. The muscles of the shoulder and arm, including the pectoralis major and minor, were much wasted; but in a marked degree less so than those of the forearm and hand. Very slight diminution of sensation. He could still lift the arm over the head. The left arm was similarly affected, but less than the right, so far as muscular atrophy was concerned—but there was numbness through the whole arm down to the fingers, accompanied with severe neuralgic pains. The trapezii, serrati postici superiores, rhomboidei, and all the long muscles of the neck and back were remarkably atrophied. The legs were wasted and weak, but the patient was able to walk, There was constipation and dribbling of the urine. He died with febrile symptoms and dyspnœa.

Autopsy.—Sections of the cord examined with the naked eye gave no distinct evidence of disease. There was a slight yellowishness of the posterior columns, and increased vascularity and thickening of the pia mater covering them. In these columns, especially in the right one, abundance of granule-cells were discovered with the microscope. The exudation was greatest in the middle and lower third of the cervical enlargement. The grey matter was hyperæmic. There was no exudation into its tissue, nor into the anterior columns. The ventricle of the cord was enlarged and distended with delicate granular nuclei.[2]

The limitation of structural changes in the cord to narrow tracts and spaces, with a healthy state of the intervening parts, and the absence of any alterations visible to the naked eye, are also strikingly illustrated in the case of Dr. P., whose spinal cord was subjected to an exhaustive examination by Mr. Lockhart Clarke.

Dr. P., æt. 65, engaged in literary pursuits, began to complain some five years before his death of neuralgic pains in the ball of the thumbs of both hands, which before long extended to the forearms and arms. After some months there was marked weakness and wasting of the muscles of the thumbs and index fingers, which also became bent inwards towards the palms. The loss of power and volume in the muscles progressed steadily, accompanied with the most excruciating pains, until his death. The right hand and arm were more profoundly affected than the left. In the later periods of the disease the pains extended to the lower limbs. The right pupil was constantly larger than the left, but the movements of the two were normal.

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[1] Gaz. Méd. de Paris, 1860. No. 32.

[2] Guy's Hospital Reports, 3d Series, vol. iv. p. 194.

The cerebellum, pons Varolii, medulla oblongata, and spinal cord, were hardened in dilute chromic acid, and sent to Mr. Lockhart Clarke. He found nothing unusual in the external aspect of the cord, neither were the anterior roots of its nerves, in any of the regions, smaller than usual to any appreciable extent. The interior of the cord, from the *filum terminale* through the whole of the lumbar and dorsal region, to the lower end of the cervical enlargement, presented no actual change of structure, either in the white or grey substance; but there was a considerable deposit of corpora amylacea round the central canal. In the cervical region, however, the case was different; for here there were decided evidences of morbid changes of structure in the posterior grey substance. These structural changes extended in a variable degree from the lower end of the cervical enlargement upwards to the third cervical nerves: they were more conspicuous at its upper than its lower part. Thin transverse sections of this part of the cord presented to the naked eye no appearance that would excite suspicion of any lesion whatever; for the morbid portions, although numerous, were small and isolated. Under a low magnifying power the posterior grey substance was seen to be interspersed with a number of unnaturally transparent streaks, patches, or spots, of different shapes and sizes. Some of these spots were seen to interrupt the course of certain nerve-fibres which extended from both the anterior and posterior cornua to the opposite side. In all the sections examined, it was around or at the side of the blood-vessels that the morbid appearances were most frequently found. The morbid spots were more numerous and extensive on the right side than on the left. The morbid spaces varied in shape, size, and relative position in the different sections. In some they appeared as mere fissures or cracks, which, under a low power, might have been considered as the result of accident, if they had not been so uniformly found in only one portion of the grey substance, and more on the one side than on the other. But when a sufficiently high power was employed, it became at once evident that they were not merely vacant spaces, but composed of a substance which differed entirely in its nature from that of the surrounding tissue. This substance had a delicate, transparent, and very finely-granular aspect. The granules were more closely aggregated toward the centre of the mass, but were generally so fine that they could not be distinctly seen under a magnifying power much less than 400 diameters. Sometimes at the edges of these morbid spaces there seemed to be a kind of transition or degeneration of the surrounding nerve-tissue into the granular substance of which they were composed. In some instances, the broken ends of nerve-fibres proceeding from the posterior roots were seen to project into the opposite sides of these spaces, across which there was strong reason to believe that they had once been continuous.

The morbid appearances gradually disappeared about the level of the third pair of cervical nerves; in the middle third of the cervical enlargement they appeared to be more extensive than elsewhere, and they disappeared on approaching the dorsal region. The sympathetic in the neck was also examined, and found normal.[1]

The peripheral distribution of the nerves to the wasted muscles was in some cases found unaltered; in other cases the nerves were found atrophied; and in one instance, examined by Frommann,[2] the nerves leading to the atrophied muscles contained fat-molecules and granular pigment.

The sympathetic in the neck was found diseased in a case examined by Schneevoogt.[3] The ganglionic cord was found extensively affected with fatty degeneration. Two similar cases have more recently been communicated by Jacoud to the Société Médicale des Hôpitaux.[4] On the other hand, the sympathetic, in two other cases, examined by Landry and Bayldon,[5] was found perfectly healthy.

PATHOLOGY.—Although defective nutrition of the muscles, ending in degeneration and atrophy, is an invariable feature of Wasting Palsy, it is evident that something more is necessary to the conception of the disease as a nosological entity. Muscles may be atrophied under a variety of pathological conditions, which are essentially distinct. Muscles may waste from want of use, as is witnessed in limbs which are temporarily kept immoveable by surgical appliances, or more permanently by anchylosis of the joints. A similar result follows severance of the connexion between a muscle and its nervous centres, especially its spinal centres; and, lastly, atrophy of muscle may follow metallic poisoning. In Wasting Palsy there is also muscular atrophy, and, so far as is known, the local changes are not essentially different from those occurring in the afore-mentioned cases; and yet how widely different is the clinical significance of the fact! In order, therefore, to obtain any clear idea of the pathology of Wasting Palsy, it is absolutely necessary to consider circumstances which are antecedent to the mere atrophy.

It must be borne in mind that the several vital endowments of a muscle may be struck with paralysis in their entirety, or singly, or in certain combinations. A muscle paralysed by a cerebral lesion loses its voluntary power, but it retains its reflex functions and its power of self-nutrition, and does not become atrophied. Other cases are known in which the peculiar "muscular sense" is lost, with preservation of all the contractile and nutritive endowments. In Wasting

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[1] Beale's Archives of Medicine. 1861.

[2] Deutsche Klinik. 1857.

[3] Schmidt's Jahrb. 1857.

[4] Nouveau Dict. de Méd. et de Chir. Paris, 1866. P. 48.

[5] See Author's Essay, p. 163, and Beale's Archives, 1861, p. 11.

Palsy, the muscle preserves its voluntary and reflex contractility, its muscular sense, and its sensitiveness to the electric stimulus; but it loses its power of healthy nutrition, and becomes degenerated and atrophied.

Pathologically, Wasting Palsy may be defined as an atrophic degeneration of certain groups of muscles, independent of any antecedent loss of mobility, or of any metallic poisoning. But the question immediately arises, whether the morbid process is primarily in the muscle itself, or in some part of the nervous system which controls its functions.

The former opinion has been adopted by Aran, Duchenne, Friedberg, Dr. Meryon, and others; it was also advocated by the present writer in his Essay on the subject, published in 1858. It must, however, be admitted that the additional facts observed since that epoch have tended materially to weaken this opinion, and to give support to the view that the primary lesion in Wasting Palsy exists in the spinal cord, or, at least, in some part of the nervous system.

The principal arguments against a nervous origin of the complaint consisted in the failure to discover, in several of the earlier post-mortem examinations, any palpable alteration in the spinal cord; and, secondly, in the want of correspondence between the range of muscles affected and the distribution of the nervous trunks. With regard to the former point, the multiplication of post-mortem examinations has very greatly increased the proportion of cases in which a lesion was discovered in the nervous system, and very much strengthened the suspicion that the earlier observations, in which the spinal cord was pronounced to be healthy, were not altogether trustworthy. The researches of Luys and Lockhart Clarke have demonstrated that profound changes in the substance of the cord may exist in detached and very limited areas, which might very easily be overlooked, seeing that it is exceedingly difficult to examine every individual section of the cord with the requisite care. Mr. Lockhart Clarke, speaking on this point, very significantly observes: "There may be very obscure structural changes in the grey substance of the cord, or perhaps only in the ganglia on the posterior roots of the nerves, that may affect the nutrition of the parts to which they are subservient, without interfering with the functions either of sensation or motion; and in cases where the lesions occur in small isolated spots, the limitation of disease to particular muscles, or even to particular fasciculi of any one muscle, could be explained, I think, by the particular nerve-fibrils within the grey substance" (Beale's Archives, 1861, p. 21).

The opinion, also, seems to be steadily gaining ground, that the nutrition of the muscles is placed under the control of a special set of organic nerves, having upward connexions with the sympathetic ganglia and the cerebro-spinal axis, which are by no means identical with the central connexions of the motor nerve-fibres of the same muscles.

Assuming the existence of such nutritive centres, all the clinical phenomena of Wasting Palsy, and the various findings of the post-mortem examinations, admit of easy explanation on the supposition that these centres, or some of their ganglionic connexions, are the primary seat of the disease. And the numerous associations and complications of the disease can scarcely be accounted for on any other hypothesis.

In considerably more than one-half of the cases now collected, and examined after death, actual disease was found in some part of the nervous system. This is a proportion which does not permit the assumption of a coincidence of two independent morbid processes. Some relation between the atrophy of the muscle and the disease of the nervous system must, I think, be admitted. Either it must be assumed that the disease of the muscle is capable of evoking disease of the corresponding nervous centre, or the converse. And although the former supposition is by no means a difficult one *à priori*, it stands on a very slender basis of fact. So far as I know, the only authenticated instance of the centripetal transmission of a morbid process along a nervous trunk is atrophy of the optic trunks after destruction of the eye. With regard to the muscles, evidence of any such transmission has yet to be given; the observations hitherto made, indeed, tend the other way. Schiff,[1] who made resections both of mixed and of purely motor and sensory nerves, found no alteration in the central portions of the cut nerves even after the lapse of a year and three-quarters. Türck[2] also examined the central origins of the nerves and their vicinities, in withered and amputated limbs, without finding appreciable alteration therein. Nor are suppurative and cancerous affections of the muscles known to be capable of transmission along the nervous trunks to the nervous centres.

The etiological conditions of some cases of Wasting Palsy, and the collateral phenomena in others, point also very strongly to a nervous origin. Several of the cases were sequential to falls or blows on the neck, or were associated with morbid growths in the spinal canal. In several well-marked cases of Wasting Palsy, also, motor paralysis, of undoubted central origin, affecting either the atrophied muscles or some other parts, preceded the atrophy. A strong impression was made on my mind by a case of this kind which fell under my notice four years ago. A young man was affected with acute paralysis of the voluntary muscles of the upper and lower extremities, unaccompanied by any wasting beyond what was due to general emaciation. After an almost total loss of motion for a period of three months, recovery set in, which, in the course of a few months, ended in complete restoration of the muscular power in all parts except the hands and feet. The muscles of these latter parts passed on to a state of characteristic atrophy, from which only partial recovery took place. Dumenil, Duchenne, and Trousseau have also published cases in which there existed motor paralysis of the tongue without atrophy, combined with atrophy without paralysis (Wasting Palsy) of the upper extremities.[1] It is easy to conceive that a morbid process in the motor centres may extend by continuity of tissue to contiguous nutritive centres (supposing such to exist), or, conversely, that disease of the nutritive centres may implicate motor or sensory centres in their vicinity, and so produce complicated clinical phenomena, analogous to those above mentioned, and which on any other supposition are very difficult of rational explanation.

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[1] Muskel- und Nervenphysiologie. Jahr. 1859. P. 122.

[2] Zeitsch. der K. K. Gesellschaft der Aerzte in Wien. 1853.

The case-history of Wasting Palsy is rich in combinations of this sort. In the pure, typical, uncomplicated cases—where atrophy of the muscles is unmixed with any degree of motor paralysis, or convulsive movements, or with numbness, or neuralgic pains—it may be assumed that the morbid process is strictly limited to the nutritive centres in the cord, or to their connexions in the sympathetic ganglia. In the complicated cases it may be assumed that the morbid process radiates into those contiguous parts of the cord which control motor and sensory functions. The question can only be finally elucidated by repeated accurate examinations of the spinal cord in complicated cases.

Prognosis.—Wasting Palsy must be counted among the most intractable diseases; and when it invades the muscles of the trunk, it almost always goes on—sometimes very slowly, sometimes more rapidly—to a fatal termination. In the partial forms—when permanent limitation of the disease to one or two members is established—life may be regarded as no longer menaced, but the usefulness of the limb, if the atrophy be complete, is hopelessly impaired. If remedial measures can be applied early, and persevered in, while the atrophy is still in progress, there is some prospect either that the advance of the disease may be permanently checked, or even that partial or perfect restoration of the injured muscles may be effected.

The gravity of the prognosis, in so far as the preservation of life is concerned, depends on the disease confining itself to the extremities, or extending its ravages to the muscles of the trunk. When the respiratory muscles are invaded, the fatal termination is not far distant.

The probability of the disease becoming generalized is greatest when the origin of it can be traced back to a hereditary predisposition. The same danger, though in a greatly inferior degree, is to be apprehended when the disease has arisen from cold, and when the lower limbs are the first attacked; also when the upper and lower limbs are *both* implicated. On the other hand, the prognosis is much more favourable when the disease is occasioned by overwork, and when it is confined to the hands and forearms.

The longer the atrophy has existed, the less is the prospect of recovery: if the disease has become stationary for a year or two, there is no chance of any considerable improvement in the condition of the muscles, but the danger to life has become comparatively small.

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[1] Bergmann, loc. cit. p. 88.

THERAPEUTICS.—In projecting the treatment of a case of Wasting Palsy, the first necessity is to ascertain, as accurately as possible, the etiological circumstances under which the disease has originated. The removal of these—supposing them to be still in operation—follows as a matter of course. Mercury and iodide of potassium have been employed with success in cases where the disease depended on a syphilitic taint. If the disease has arisen from overtasking any set of muscles, these must be allowed to remain at rest.

The direct treatment embraces the employment of hygienic means —baths, methodical exercise, change of air, &c.—and the employment of galvanism and frictions to the affected muscles. Remak strongly advocates the use of the constant galvanic current applied to the spinal cord—especially the cervical portion.

Thermal and sulphur baths have been highly recommended by a number of writers. Wetzlar has especially called attention to the beneficial effects of the waters of Aix-la-Chapelle. Cold baths are objectionable.

The most effective remedy in Wasting Palsy is, undoubtedly, galvanism. Numerous observations attest its value when applied locally to the affected muscles. After a very considerable experience of its employment, I am convinced that it very rarely fails of some good effect when perseveringly applied. This effect is too often temporary: too often also it is found difficult to keep up the treatment with the requisite regularity for a sufficient length of time. In some cases marked improvement in the power and bulk of the muscular masses was witnessed; in others, the disease, previously progressive, was brought to a standstill. In the case of a man, still under observation, suffering from atrophy of the muscles of the thighs and upper arms, and of the erectores spinæ, which had been steadily progressive for twelve months, the daily application of the secondary current arrested the disease completely. The arrest has now continued for more than six years.

Duchenne gives the following directions for the employment of galvanism:—"Every muscle ought to be Faradised in a special manner, according as it has suffered more or less in its electric contractility and nutrition. Thus the more a muscle is atrophied and its contractility diminished, the longer it should be subjected to the stimulation, the more intense should be the current, and the more rapid its intermissions. And this strong current and quick intermissions are the more necessary, according as the sensibility of the muscle is more benumbed. But when the sensibility is seen to return, it is prudent to diminish the intermissions and abate the intensity of the current, and even to abridge the number of sittings, lest there be provoked unmanageable neuralgia, and, which sometimes has arisen, inflammatory accidents. During the Faradic treatment, I have excited the muscular sensibility, as much as possible, by rapid intermissions, inasmuch as I have found this the most effective means of reacting on the nutrition of the atrophied muscles. Sittings of too long duration fatigue and even exhaust the muscles, just as forced exercise induces atrophy, instead, like moderate exercise, of favouring nutrition. I believe that no sitting should be protracted beyond ten or fifteen minutes, at the most. I rarely give more than one minute to each muscle. To prevent weariness, and a bruised feeling, that sometimes follows the application of electricity, I pass rapidly over the muscles, taking care to return to each of them several times during the same sitting, so as to leave a short interval of repose between each excitation."[1]

The secondary symptoms—cramps and neuralgic pains—are most effectually subdued by warm baths, temporary rest in bed, and anodynes. The hypodermic injection of morphia has, in my hands, been followed by the happiest effects in relieving the excruciating neuralgia which is not unfrequently associated with this disease. One of my patients, thus afflicted, is in the habit of having half a grain of morphia injected early in the morning, when the pains are severe. Such an injection enables him to pursue his employment through the day in comfort—a result which he fails to attain by any dose of the same remedy internally administered.

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[1] De l'Electrisation localisée, p. 702.

METALLIC TREMOR. TREMBLEMENT MÉTALLIQUE.

By William Rutherford Sanders, M.D., F.R.C.P.

Synonyms.—Tremor Metallurgorum; Paralysis Agitans Metallica; Rheumatismus Metallicus (Schönlein); Metallic Shaking Palsy; The Trembles.

1st. Mercurial Palsy or Tremor; Mercurial Shaking Palsy; Mercurial Trade Disease; Tremor ab Hydrargyro; Paralysis Agitans Mercurialis; Tremblement Mercuriel; Tremblement des Doreurs; Mercurial-Zittern.

2d. Lead Tremor or Shaking Palsy; Tremor Saturninus; Paralysis Agitans Saturnina; Saturnines Zittern.

Definition.—Metallic Tremor is a species of paralysis agitans, caused by the slow poisonous action of certain metals, particularly mercury and lead. It consists of spasmodic tremors with diminished muscular power, occurring in various parts of the body.

1. *The Mercurial Tremor or Shaking Palsy,* being the form best known and most important, will be first described.

Causes.—*Exciting.*—The chief source of this disease is the inhalation of mercury in a state of vapour, this metal being volatile at nearly ordinary temperatures (68° to 70° Fahr.). By some authors this has been regarded as the only mode of origin; but it is certain that the introduction, of mercury by the skin, either in consequence of manipulating the metal, or of prolonged friction with mercurial ointment, has sometimes brought on the peculiar tremors; and the same effect has also resulted, in a few instances, by absorption, from the intestinal canal, of mercurial preparations administered medicinally. The principal sufferers from the disease are accordingly:—1st. The workmen employed in the quicksilver mines, especially when fire is used in the reduction of the ores. 2d. Water-gilders (who plate with gold dissolved in mercury), looking-glass silverers, barometer makers, workmen in chemical manufactories, where mercurial preparations are made, button and toy gilders, furriers, and others whose business exposes them to contact with mercury.[1] 3d. Persons using mercury medicinally. In former times, the Iatraliptæ, an inferior class of surgeons, who practised as mercurial anointers or rubbers, without protecting their hands, were frequently subject to tremors which sometimes proved incurable. A similar instance is recorded recently. Dixon, the anatomy porter of the Irish College of Surgeons, "who at one time rubbed in immense quantities of mercury for the cure of venereal among the *Mohawks,* or swells of the day," was subject for thirty years to mercurial stammering (psellismus mercurialis).[2] Syphilitic patients, after long courses of mercurial treatment, especially by friction, often suffered severely from the trembles.[3] On the other hand, the internal use of mercurial medicines alone very rarely gives rise to the tremors; nevertheless undoubted examples of this kind have been observed even in recent times, both in venereal and in other cases.[4] In the present day, there is little risk of tremors originating from excess in either the external or internal medicinal use of the mineral, but the possibility must not be overlooked. 4th. Persons are sometimes accidentally exposed. In 1810, the *Triumph,* man-of-war, took on board a cargo of mercury, saved from a wreck. In consequence of the bladders bursting, in which it was held, the mercury spread through the ship, and in the space of three weeks, "two hundred men were afflicted with ptyalism, ulceration of the mouth, partial paralysis in many instances, and bowel complaints."[5] In 1803, a fire broke out in the quicksilver mine at Idria, near Trieste, and about nine hundred persons in the neighbourhood were attacked with nervous tremblings.[6] Medico-legal questions have also risen as to the alleged deleterious effects of emanations from workshops where mercury was used.[7]

In a few instances a single strong exposure has been known to cause the tremors;[8] but usually a prolonged and habitual contact, for months or years, is required, under conditions which favour the development of the disease.

Predisposing Causes.—The circumstances which dispose to the disease or aggravate it, are:—1st. Bad ventilation; 2d Cold and damp weather (hence the tremors are worse in winter, in consequence of the low temperature and close confinement); 3d. Defective cleanliness; 4th. Intemperance: 5th. Violent emotions (a fit of passion has sometimes originated an attack of tremors suddenly); 6th. Idiosyncrasies must be taken into account. Certain constitutions are more susceptible than others to the mercurial poison. The same exposure which in some individuals affects the mouth, producing salivation and ulcerations of the gums, without tremors, will, in others, cause tremors without salivation. 7th. The mode of application has considerable influence. As a rule, inhalation of mercurial vapour is followed by tremors; inunction or internal medicinal use, by salivation.

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[1] Ramazzini, De Morb. Artif. caps. i.—iii. 1717; Patissier, 1822; Thacrah, 1832; Darwall in Forbes' Cyc. Pract. Med. i. 151, 1833; Tardieu, Dict. d'Hygiène, 1852; Whitley in Sixth Report of Med. Officer of Privy Council, 1863, p. 358.

[2] Mapother, Mercurial Trade Disease, Med. Press and Circular, i. 531, May 23, 1866.

[3] Hutten, De Morb. Gall.; Fernelius de Luis, Ven. Cur. c. vii. p. 234, 1656; Ramazzini, l. c.

[4] Colson, Arch. Gén. de Méd. xv. 338, 1827; Lancet, ii. 1838–9, p. 767.

[5] Burnett, Phil. Trans. 1823, Pt. ii. 402. The *Phipps* schooner, which assisted, was similarly affected: Ed. Med. and Surg. Jour. vi. p. 513, 1813.

[6] Murray's Handbook to S. Germany, 9th edit. 1863, p. 400.

[7] Chevallier, Annal. d'Hygiène, xxv. 388, 1848; Orfila, Toxicologie, 4th edit. 1843, i. p. 593.

[8] Christison on Poisons, Merc. Tremor, 4th edit. p. 418, 1845.

Description.—Previously noticed by several writers, and especially by De Haen,[1] the tremulous mercurial disease has been most fully described by Mérat,[2] as observed among the water-gilders of Paris. Less complete accounts have been given by various authors of the disease among workmen in other countries, and as it affects the quicksilver miners at Almaden and Idria.[3]

Symptoms.—1*st Stage. Simple Tremors.*—The commencement is sometimes sudden, but most frequently the disease comes on gradually. The upper extremities are nearly always first affected. The patient finds his hands and arms getting weak, unsteady, and less under control; they vacillate and tremble whenever they are used. He can do coarse work, but nothing requiring precision. The attempt to seize or hold anything increases the trembling. At the same time numbness or formication is sometimes felt in the hands or feet, and occasionally pains in the joints, particularly the thumbs, elbows, knees, or feet. These simple tremors are very common among quicksilver miners and water-gilders. They are not so severe as to prevent work altogether, and by judicious means they may be kept from increasing.

2*d Stage. Convulsive Tremors.*—If the patient continues or increases the exposure, or becomes more susceptible to it, the trembling augments in intensity till it becomes convulsive or spasmodic in character. Muscular subsultus occasions vibration and jerking of the hands and arms. The tremor is easily excited either by exertion or emotion, and once begun cannot be stopped for some time. The voluntary acts also become spasmodic as well as tremulous, and are accomplished by interrupted violent starts, like the movements in chorea. In bending the arm, for example, the flexion cannot be done by a single continuous contraction, but takes place by two or three jerks. The tremulous hand cannot be directed with precision, but is projected beyond or beside or away from the object; it soon becomes unfitted for work, and can scarcely convey food or liquids to the mouth. As Dr. Pope tells of a miner at Friuli, "he could not with both his hands carry a glass half full of wine to his mouth without spilling it, though he loved it too well to throw it away."[1] The convulsive nature of the movements depends greatly on the predominance of the flexors over the extensors; so that when a patient has seized an object, he often cannot let go his grasp. At this period, the patient is usually obliged to discontinue work, and after an interval of rest, steadiness may still be completely restored. But if he persist, or resume his employment too soon, the tremors become greatly aggravated, and extend by degrees over the whole body. The legs begin to shake, especially at the knees, and in walking they tremble and dance as if hung upon wires. The lips, tongue, and jaws are in tremulous vibration, and speech is hurried, staccato, and stammering, becoming at last unintelligible (psellismus mercurialis). The head oscillates, shaking, or nodding; and sometimes the features are distorted by spasmodic grimaces; the eyeballs alone are unaffected in their movements. Mastication is impeded. Finally, the tremulous subsultus appears in the muscles of the trunk, and the respiratory movements are convulsive and attended with dyspnœa. Tonic spasms also occur in the affected parts, and are frequently attended by pains, to which the Spanish miners of Almaden give the name of "calambres," *i.e.* cramps. These pains are sharp and lancinating, and sometimes of intolerable intensity; they are not always in proportion to the muscular contraction.[2]

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[1] De Haen, Ratio Medendi, Pt. iii. c. 28, 1761.

[2] Mérat, Mém. sur le Tremblement des Doreurs, &c.; Appendix to the Traité de la Colique Métallique, Paris, 1812; also in Dict. des Sc. Méd. xxx. 232, 1818, and lv. 521, 1821; Bateman, Ed. Med. and Surg. Jour. viii. 376, 1812; Mitchell, Lond. Med. and Phys. Jour. 1831, p. 394; Bright, Med. Rep. ii. 495, 1831; Stokes, Ryan's Lond. Med. and Phys. Jour. v. 519, 1834; Lancet, ii. 1853, pp. 231 and 317; Med. Times, ii. p. 578, 1853; Marshall Hall, Watson, Romberg, Valleix, Falck in Virch. Handb. der Spec. Path. u. Ther. I. iii. 136, &c.

[3] Jussieu, Mém. de l'Acad. Roy. des Sciences de l'Année 1719, p. 357, &c.

When the tremors attain their greatest intensity, they amount to a kind of convulsion, and the patient presents a most pitiable aspect. In constant tremulous commotion, tottering, trembling, shaking, and stuttering, he is powerless to execute any combined movement; he cannot walk, or speak, or chew; he dares not touch any object for fear of breaking it or letting it fall; on raising his agitated hand, with food, to his mouth, he misses his aim and inflicts involuntary blows on his face. He must be fed and clothed like a child.[3] Some unfortunates, deprived of assistance, have been known to creep on all-fours, and seize their food with the lips, like the lower animals. Unless in the very worst cases, however, whenever the body is supported, sitting or reclining, the tremors gradually subside, and soon cease altogether, and they do not return until excited in consequence of some voluntary movement or mental emotion. During sleep, they remain in entire abeyance. The patient is thus allowed time for repose and recovery. But in the most advanced cases, the subsultus takes place even when the body is reposing, so that the involuntary shaking of the head on the pillow has prevented sleep.[1] In the tremulous parts, the muscular strength is diminished (paresis), but there is no interruption to the conduction of the stimulus of volition (paralysis). The sensibility is not impaired.

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[1] Phil. Trans. i. p. 21, 1665.

[2] Tardieu, Dict. d'Hygiène, ii. 481, 1854, who quotes Roussel, Lettres Méd. sur l'Espagne, Union Méd. for 1848–9; Ed. Monthly Med. Jour. Retrospect for 1843, p. 254.

[3] De Haen's description is graphic. Case 2: "Deaurator, 25 annorum, horrendo artuum omnium, maximè superiorum . . . vexatus . . . ita ut nihil laboris ultra perficere, ut nec comedere, bibereve solus, nec loquens amplius intelligi potuerit. Nutriendus, vestiendus et infantis instar, alvum urinamque positurus, adjuvandus erat; dolorum cæterum immunis." It is satisfactory to add, "Virtute electreâ trium septimanarum spatio adhibitâ, perfectissimè convaluit, ita ut ipsi, sive in motu, sive in loquelâ, ne vel minimum quidem desit." (Loc. cit.)

Concomitant Symptoms.—The condition of the other functions, accompanying the disorders of the nervous system just described, indicates the presence of the mercurial cachexia. At the beginning this is slight and unimportant. The skin exhibits a sallow, brown, or earthy tint; it is dry and sometimes rather warm; the expression is sometimes animated, at other times languid; there is little or no emaciation, which, indeed, does not appear till the disease is of long standing. The digestive functions are unimpaired; there is no colic, the abdomen is soft and of ordinary volume, and the urinary and alvine excretions are natural. But as the tremors become more severe, the appetite diminishes, and it ultimately ceases altogether; the tongue becomes white and pasty, but without bad taste, and gas accumulates in the intestines. The respiratory organs are natural, till dyspnœa and asthma arise, from the respiratory nerves being involved in the tremors. The pulse is usually at first strong and slow, as in metallic colic, but it may afterwards become small and weak; sometimes it is accelerated.

But sooner or later signs of general mercurialism usually make their appearance, especially salivation, loose teeth, inflamed and ulcerated gums, aphthæ, fetid breath and sweat, swelling of the parotids, and a pustular eruption over the body. These symptoms occur early, and are particularly obstinate in workmen who take their food in the workshops or mines, and who are not careful to use ablutions, and change their clothes and shoes. Attacks of excited circulation (erethism) are frequent in the early stages; and in the later, anæmia, emaciation, and great debility. If we except the cachectic symptoms just described, complications are rare in the course of mercurial tremors. The colic, which is sometimes observed, depends upon lead which is mixed with the mercury, or has been used along with it.

3d *Stage. Mercurial Tremors, with Affection of the Brain.*—The tremors are not of themselves dangerous to life, but in the advanced stage they are often accompanied by serious cerebral disorders, as headache, loss of memory, loss of consciousness, sleeplessness, delirium, epilepsy. These symptoms would soon end fatally were it not that generally their gravity compels the sufferer to desist from his employment; and by this fortunate interruption, recovery usually takes place even from this dangerous condition. Indeed, the disease when subjected to treatment is rarely fatal.[2] Some inveterate cases prove incurable, or are succeeded by motor paralysis, but it is only in those instances where the noxious exposure is obstinately persisted in, notwithstanding repeated attacks of increasing severity, that death finally takes place, accompanied by symptoms of profound mercurial cachexia, and especially extreme marasmus and exhaustion.

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[1] De Haen, loc. cit. case 7.

[2] See fatal case from "general failing of the vital powers." Lancet, 1839-40, ii. p. 588, and Guy's Hosp. Rep. 1864, p. 175.

But although mercurial tremor by itself is not directly fatal, and is a curable disease when submitted to proper treatment, yet, under the circumstances in which workmen were placed till within a recent date, the effects of the mercurial poison, taken as a whole, were most disastrous, and the mortality in certain employments was excessive. In Paris, in 1821, it is stated that the looking-glass manufacturers could not remain at the trade above eight or twelve years. When necessity compelled them to persevere too long, their faces became pale, with an expression of intoxication, their intelligence and memory gradually failed, they fell into a kind of idiotcy, and after lingering in this state for some years, they died of consumption, or were struck with apoplexy.[1] In 1847, Dr. Sanderet reports that the trade of water-gilding at Besançon, where it was extensively carried on, was most injurious to health,[2] the mortality among the workmen being enormous, and due chiefly to phthisis. Fortunately these trades, conducted under better hygienic conditions, or by means of new processes, are either entirely innocuous or are much less injurious at the present day.

The condition of the quicksilver mines was, in ancient times, most dangerous.[3] At a comparatively recent date, when Jussieu visited Almaden, in 1719, he found that the free miners, who adopted proper precautions, preserved their health and lived like other men, but the convicts and slaves who took no care suffered severely, and fell victims to disease. In 1848-9, there were no slaves nor convicts in those mines; but it was observed that the native miners, who knew the risks and avoided them, were little affected, while the poor labourers from a distance, careless and dissipated, experienced the most disastrous effects. The average number of workmen was 3,911; of these forty-eight were "calambristes" (in the second stage of mercurial tremors), half of which number died within the year, and the other half remained unfit for work in the mines. Besides this, there were two deaths from accidents, three mutilations, and thirty-nine injuries more or less serious. And although many workmen do not fall victims to the mercurial poison, none of them entirely escape its action.[4] At Idria, although the hygienic conditions of the place are in other respects highly favourable, it is stated that the whole population is subjected to the influence of mercury, not the workmen at the mines only. The annual mortality is 120 out of 4,500 inhabitants. The workmen exposed directly to the action of the metal suffer severely. In 1856, 122 out of 516 were seriously affected.[1] Information is wanting in regard to the quicksilver miners in California and Australia.

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[1] Burdin, Art. Tain, Dict. des Sc. Méd. 1821, liv. 276.

[2] "Une des industries des plus fatales à la santé." (Annal. d'Hygiène, 1847, xxxviii. 457.)

[3] At Idria, in 1665, Dr. Pope says, "All of the miners in time (some later, some sooner) become *paralytick* and dye *hectick*." (Phil. Trans.) Also Dr. Edward Brown, in Phil. Trans. Dec. 13, 1669.

[4] Tardieu, loc. cit., and Roussel. In the enclosure called Brutrones, where the furnaces are situated, the animals which are allowed to graze there are liable to Mercurial Tremors. (Edin. Month. Med. Jour., Retrospect for 1848, p. 255.)

COURSE AND PROGNOSIS.—Mercurial Tremor is essentially a chronic and protracted disease. It runs a uniform course. Once begun, if the exposure is persevered in, the symptoms gradually get worse; the tremors become more intense, are accompanied by spasms clonic and tonic, and spread over the whole body. But if the patient be removed from the exciting cause, exposed to fresh air, and placed under suitable treatment, amelioration soon begins, and, after a few weeks or months, perfect steadiness may be restored. The prognosis, therefore, is generally favourable, provided the patient can avoid the contact with mercury. The prospect of cure, and the time required for it, will depend on the severity of the symptoms, and especially on their duration previous to treatment, on the age of the patient, the presence of serious cerebral symptoms, and the degree of mercurial cachexia which may accompany the tremors. The affection is most frequent probably in middle life (thirty to forty); it is more severe in old people. If taken at an early stage, twenty days may effect a cure, but in a confirmed case usually from two to seven months, sometimes a year or more, are required. When the tremors are spasmodic and generalized, the cure is tedious and imperfect, some tremor of the hands nearly always remaining permanently. The upper extremities, which are the parts earliest and most severely affected, resist cure the longest. It is seldom that the tremors are persistent and irremediable, and, as already stated, fatal results only ensue in consequence of general cachexia or phthisis or apoplexy, the effects of an unhealthy constitution, or of unpardonable neglect, or of obstinate persistence in exposure to the poison.

After a first seizure relapses are frequent, and usually of increasing severity. If the patient, in spite of due care, is still subject to attacks, he ought to change his employment. Some constitutions, peculiarly sensitive to the poison, are unfit for any trade requiring the use of mercury.

DIAGNOSIS.—The symptoms and the cause distinguish Mercurial Tremor readily from other diseases. It could only be confounded, 1st, with chorea, or St. Vitus's dance, which it resembles in the jerking nature of the movements, but it differs by the presence of tremors; 2d, with idiopathic paralysis agitans, with which it is identical as regards the character of the irregular movements (viz. tremors and jerking), but it is distinguished from it by the exciting cause (mercury), and by the concomitant symptoms of mercurial poisoning. In addition, the speech (tongue and jaws) are much sooner, more invariably, and more characteristically affected in the mercurial disease than in idiopathic shaking palsy. The loss of memory and consciousness, and other cerebral symptoms also, are peculiar to the mercurial disease. On the other hand, the irresistible tendency to walk or run forwards, which marks the paralysis agitans festinans, is not met with in the Mercurial Tremors; the only disturbance of equilibrium in the latter is that which results from debility, tremors, and spasmodic jactitation.[1] 3d. It is not likely, with ordinary care, to be mistaken for delirium tremens, or alcoholism.

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[1] Med. Times and Gazette, xxxix. p. 616, 1859, and Gaz. Hebd.

PATHOLOGY AND MORBID ANATOMY.—The disease being rarely fatal, the information in regard to the morbid anatomy is scanty. In a recent case,[2] Dr. Alfred Taylor found the brain and spinal cord, the muscles, lungs, heart, liver, and kidneys, in appearance quite healthy. On chemical examination of the brain, liver, and kidney, minute globules of metallic mercury were obtained, in largest proportion from the kidney. The spinal cord and medulla oblongata are doubtless the seat of the principal morbid action, just as in idiopathic paralysis agitans. The molecules of mercury entering probably into combination with the nervous substance, seem both to irritate and partially to paralyse the nervous centres of motion, while they leave the apparatus of sensation intact.[3] The opinion which some authors entertain, that the morbid lesion has its seat in the muscles and not in the nerve-centres, is insufficient to account for the spasmodic and variable nature of the phenomena, and is inconsistent with the cerebral symptoms which ultimately become developed.

The *treatment* is twofold, preventive and curative.

Prevention is accomplished, 1st, by limiting the exposure to a short period at considerable intervals, or by adopting various contrivances which remove the mercury from contact with the operator. In the time of Pliny[4] the workmen protected their faces with masks of loose bladder skin, sufficiently transparent to admit of being seen through. Masks of glass were afterwards substituted. Sponges over the mouth, and various kinds of respirators, have also been proposed. But the chief improvement has taken place in recent times by the better construction and ventilation of the workshops, and by the introduction of flues and chimneys, which carry off the mercurial vapours by a powerful draught out of the apartment, while the workman is further protected by a glass sash interposed between his face and the stove where the mercurial vapours are disengaged. To D'Arcet's[5] draught chimney for this purpose the French Academy of Sciences, in 1816, awarded the prize founded by M. Ravrio, who had made his fortune as a manufacturer of gilt bronzes, and was anxious to obtain some means for protecting workmen from the risks of the employment. Mérat bears testimony to the efficacy of D'Arcet's chimney in warding off the tremors, to some extent. Similar flues and stoves have been used in this country;[1] but none of these inventions have proved successful in entirely preventing the disease.—2d. On the part of the workmen, regular habits, personal cleanliness, change of clothes, frequent ablutions, and the practice of never eating in the mine or workshop, or with unwashed hands, are essential. Intemperance invariably predisposes to or aggravates the disease. Melsens observed that workmen who used much salt with their food are less liable to suffer from Mercurial Tremors, and he also recommended the iodide of potassium as a powerful preservative. Of late years in this country Mercurial Tremor has greatly diminished in frequency, and under proper hygienic rules would probably cease altogether, at least in its more aggravated forms. Water-gilding, the most dangerous kind of mercurial trade, has been now almost altogether superseded by electroplating, which is completely innocuous. Looking-glass silvering, when conducted in large well-ventilated apartments, with means for preventing the diffusion of the metallic dust, is also quite safe, if the workmen are employed only at intervals, and are careful and temperate. But when these conditions are not attended to, and especially if the men are kept too continuously at work, slight tremors soon make their appearance, and severe cases occasionally happen.[2] According to Dr. Whitley's report,[3] the number of persons affected in England and Wales appears to be small, and the cases, for the most part, slight. The same statement may be applied to Scotland and Ireland. The condition of the quicksilver mines is probably still one of considerable danger to health and life. But full and accurate information is wanting in regard to the amount of sickness and mortality among the miners, and the means used for their protection.

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[1] De Haen's fourth case presented considerable disturbance of the balance in locomotion. "Adeo difficulter ingreditur, ut sæpius humi concidat, dumque corpus pronando sustinere se nititur, vi quasi supinatur." (Loc. cit. p. 230.)

[2] Guy's Hosp. Rep. 1864, x. 176; Lancet, 1839-40, ii. p. 589.

[3] Jussieu's idea of the pathology is curious. The tremors, he says, are "les tristes effets du séjour du sang dans les vaisseaux du cerveau, dévenus variqueux par le poids de quelques particules mercurielles, qui y ont séjourné." (Loc. cit. p. 360.)

[4] Hist. Nat. xxxiii. 40.

[5] Mémoire sur l'Art de dorer le Bronze; Paris, 1816. Dict. des Sc. Méd. 1818, xxvii. p. 299.

The *Curative treatment* consists, first, in complete removal of the patient from his noxious employment; next, in change of dress, ablution, exposure to a free atmosphere of moderate temperature, and the administration of a nutritious tonic diet. The objects of treatment then are, 1st, to eliminate the mercury from the system by the secreting organs. *Sudorifics* [4] have been much used for this purpose: acetate of ammonia, Dover's powder, guaiac, sarsaparilla, sassafras, &c.

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[1] Darwall in Forbes' Cyc. Pract. Med. 1833, i. 157.

[2] See recent cases, Scott Orr in Glasg. Med. Jour. i. 37, May 1866, and Mapother, loc. cit. Also Taylor, loc. cit.

[3] Sixth Report of Med. Officer of Privy Council for 1863, p. 22; and 1864, p. 358.

[4] Jussieu remarks (1719) that at the mines of Almaden the medical treatment differed from the usual practice then in vogue of purging and bleeding, and consisted simply in exposing the patients to the free air, and administering absorbents, as hartshorn, ivory, or crab's-eyes; and what is singular (he adds) the treatment succeeds almost always in temperate subjects and those who abstain from wine, whilst those who indulge perish without resource. (Loc. cit.)

Sulphur has been regarded by some as specific; warm and vapour baths, or sulphurous baths, &c. are always employed. Neufchâtel had a reputation for the successful treatment of water-gilders' palsy, chiefly by means of vapour baths.[1] *Diuretics* have become favourite remedies, and especially the iodide of potassium, since Melsens[2] brought evidence to show that this salt has the power of redissolving the mercury contained in the tissues and eliminating it by the urine, in which its presence may be detected chemically.[3] The caution must be observed not to give the iodide so largely as to disengage an excess of mercury at once within the body. Various other diuretics, common salt, bitartrate of potash, &c. may be employed. Purgatives are also useful. 2d. Another indication is to soothe and strengthen the nervous system, by means of *antispasmodics, narcotics, tonics,* and *stimulants.* Steel and quinine, singly or in combination, are especially serviceable. Opium is useful. Nitrate of silver has also been recommended. Stimulants, particularly alcohol, wine, &c. exert a powerful immediate effect in arresting the tremors: hence workmen are apt to resort to them to steady their hands; but when the immediate effect is over, they aggravate the tremors. The most beneficial stimulants are *electricity* and *galvanism,* which have afforded very satisfactory results.[4] De Haen's cases were cured, some of them rapidly, with electricity as the only remedy applied.

2. *Lead Tremors; Tremor Saturninus; Paralysis Agitans Saturnina.*

Mérat denied that any other metal than mercury could give rise to tremors, and, with few exceptions,[5] other writers appear to be of the same opinion. In regard to artisans using lead, this view is probably correct; but the case is different with miners exposed to the vapours of the metal. Brockmann,[6] in particular, from his experience in the Harz Mountains, has described a species of lead tremors, which affects the miners there, and which is almost identical with the mercurial shaking palsy, consisting, like it, of oscillating spasmodic contractions of the muscles, and consequent tremulous motion in various parts of the body. His account includes two forms, the local (partialis) and the general (universalis), both the result of an affection of the nervous centres by lead.

[1] Sandaret, Ann. d'Hygiène, 1847.

[2] Annal. de Chimie et de Physique, 1849, xxvi. 215, and transl. in Brit. and For. Med.-Chir. Rev. for Jan. 1853, p. 217.

[3] Schneider of Vienna controverts the assertions of Melsens, Ed. Med. and Sur. Jour. 1861-2, p. 394.

[4] De Haen, loc. cit. Gull Guy's Hosp. Rep. 1853, viii. p. 136.

[5] Percival, Ed. Med. and Surg. Jour. 1813, ix. 62, ascribed tremors rather to lead than to mercury!

[6] Die Metallurgischen Krankheiten der Oberharzes, 1851, p. 282; Schönlein, Allg. und Spec. Path. und Ther. 2 Thele, p. 191 (St. Gallen, 1841); Falck, in Virch. Handb. d. Spec. Path. und Ther. ii. 1 Abth. 517-8, 1855. Wilson, in Edin. Essays Physical and Liter. 2d edit. 1771, p. 517, in describing the disease called *Mile-Reek* among the miners at the Lead hills, mentions that the "extremities tremble and are convulsed." Sauvages, Nosol. Meth. 1768, p. 558, Tremor Metallurgorum.

SYMPTOMS. 1*st. Tremor Saturninus partialis.*—As a rule, the upper extremities are alone affected. The arms and hands are in continual vibration, more or less, greatest when any powerful effort is made, or during emotion. With this there is often associated a peculiar nervous tremor of the lips (musc. orbic. oris) and angle of the mouth (levator anguli oris), like that observed when a shy sensitive person opens his lips. The local tremor usually follows violent and persistent attacks of lead colic, especially in highly nervous subjects, or in those exhausted by previous disease. It disappears mostly in a few days, but is apt to return when the exciting causes are renewed. Under very unfavourable circumstances, however, it may increase and extend into the general form of the affection.

2*d. Tremor Saturninus universalis.*—In this the tremors are not confined to the arms, but appear in the legs and muscles of the head and trunk. The patient presents a peculiar and pitiful aspect. When at rest, his back is bent like an old man's, his head is bowed, and the chin falls upon the breast; in walking, the legs are rotated tremulously as in paralysis agitans. In advanced cases the jaws, and indeed all the muscles of the head and body, are the seat of the uncontrollable tremors which characterise the disease.

CAUSES.—The general tremor never results except from deeply-rooted lead-poisoning. It is preceded by repeated outbreaks of the severer forms of lead disease, and progresses hand in hand with the lead cachexia. All the causes of the cachexia predispose to it, and the tendency is increased by an excitable nervous constitution, together with lax fibre and weak muscular system. Age has no appreciable influence. The saturation of the system with lead is the only exciting cause of the disease.

The COURSE of the general disease is chronic and very protracted. Months or even years elapse before permanent improvement takes place. Often a radical cure is impossible. Frequently, also, the tremor becomes associated with some form of anæsthesia, or ends in complete paralysis.

The PROGNOSIS is consequently almost always unfavourable. Only when the disease is partial, and follows a violent colic, or an acute attack of convulsions, can a speedy favourable issue be anticipated. Under all other circumstances, lead tremor is a most serious affection, and is apt to be the precursor of more sudden and dangerous disorders, such as paralysis or cerebral disease.

PATHOLOGY AND MORBID ANATOMY.—No specific lesion has yet been pointed out in the brain or spinal cord. The affected muscles have been found altered, but this is of subordinate importance. The pathology is doubtless analogous to that of mercurial tremor, and idiopathic paralysis agitans.

The DIAGNOSIS is sufficiently determined by the symptoms of the lead cachexia which accompany the tremors, and by the absence of any source of mercurial poisoning.

The TREATMENT must be directed to neutralize the lead poison, and to strengthen the nervous centres, which are the chief seat of the disease. The energetic employment of *sulphur baths,* cold water *douches* to the spine, and the internal use of *nervine tonics,* nux vomica or strychnia, valerian, quinine, &c. are the appropriate means. Sudorifics, sarsaparilla, &c. have also been used, and Melsens recommends the iodide of potassium on the same grounds as in the case of mercury. Brockmann states that he never concludes the treatment without the persevering use of baths of aromatic herbs, and the cold plunge bath, as well as the internal administration of chalybeates. In obstinate cases electricity and galvanism must be resorted to, but frequently without much success. The most essential part of the treatment consists in the removal of the patient from his unhealthy employment, and placing him in free pure air, with a nutritious animal diet, and a moderate allowance of good wine and beer.

In poisoning with arsenic, zinc, or bismuth, tremors frequently occur, but they only form part of a general group of symptoms, and do not require particular description in this article.

B. Partial Diseases of the Nervous System.

1. Diseases of the Head.

a. Meningeal Diseases:—

Simple Meningitis.
Tubercular Meningitis.
Chronic Hydrocephalus.
Meningeal Hæmorrhage.
Adventitious Products.
Congenital Malformations.

b. Cerebral Diseases:—

Congestion of the Brain.
Cerebritis.
Softening.
Adventitious Products.
Cerebral Hæmorrhage.
Abscess.

SIMPLE MENINGITIS.

By J. Spence Ramskill, M.D.

Definition.—By Meningitis is generally meant inflammation of the pia-mater and arachnoid. Inflammation of the dura-mater is described separately. Attempts have been made to separate inflammation of the arachnoid from that of the pia-mater, and some, as Lallemand, Parent-Duchâtelet, and Martinet, have even gone so far as to apply the term Arachnitis to inflammation of the arachnoid, on the ground that the serous membrane was the one chiefly affected. But an analysis of the cases given by the very advocates of that opinion shows most conclusively that the pia-mater is in all cases affected, and always bears more marked evidences of inflammation than the arachnoid. There is no symptom which, during life, could help to distinguish between inflammation affecting the pia-mater and inflammation involving the arachnoid alone; and as the treatment in either case would be the same, there would be no practical advantage gained by such a distinction. Cerebral Fever is a name given by Trousseau to various acute affections of the head in children, amongst which he includes Meningitis. Meningitis may be primary or secondary; uncomplicated or complicated; acute or chronic.

Acute Meningitis. Symptoms.—In some rare cases, certain prodromata precede the invasion of the disease, in the shape of slight but increasing pains of the head, irritability of temper, sleeplessness, and general malaise. But, as a rule, the invasion of the disease is decided, and from the outset its gravity is not to be mistaken. Its course may be divided into three stages:—1st, a period of excitement; 2d, a period of transition; 3d, the stage of collapse. These three stages are not always present, nor are they always distinctly marked. When the disease is very violent, the first stage may rapidly pass into the third, or comatose period. In old and feeble people the first stage may either be absent altogether, or be so little marked as to escape observation. Again, the third stage may be absent, from life being abruptly cut short by violent general convulsions in the second period; and lastly, the first and third stages may coalesce during the transition from one to the other, and may present mixed phenomena of delirium alternating with coma.

Stage First. Period of excitement.—A well-marked rigor, with pallor of the surface and cutis anserina, opens the scene, and is very quickly followed by intense febrile reaction. In very young children a paroxysm of general convulsions may be the first symptom; in adults, however, convulsions are the exception. The fever is very high; the skin is hot and dry; the pulse frequent, sharp, and hard; the face flushed, particularly about the malar bones: sometimes it is alternately flushed and pale. The eyes are glistening, the conjunctivæ injected; the pupils in this stage are usually contracted; there is photophobia; and, in order to keep the light out, the patient keeps his eyes firmly closed, and resists all attempts at opening them on the part of the practitioner. There is sometimes strabismus on one or both sides, particularly in children. Acoustic dysæsthesia distresses the patient: the least sound, the lightest footstep about the room, gives him pain. Cephalalgia of the most acute character sets in from the first. It is referred to the forehead, vertex, temples, or occiput, or to the head generally: pressure on the scalp increases, and movement intensifies it; hence, in order to prevent his head from moving, the patient holds it between his hands. Sensorial impressions of light and sound also exaggerate it. The pain is continuous, but presents also frequent exacerbations, during which the patient, especially if a child, utters a peculiar, loud, piercing cry. The headache may precede the other symptoms for a day or two, or for a few hours only, or it may appear simultaneously with them. It is the most striking symptom of the disease; it is present in nearly every case, but not in all: and Andral has related in his "Clinique Médicale," cases in which it was absent from first to last; and, in one of these, sero-purulent effusion was found in the lateral ventricles after death. The intensity of the pain does not bear any relation to the stage of the inflammation and the nature of its products. Thus, it has been found as severe in cases where a post-mortem examination disclosed mere injection and increased vascularity of the meninges, as in cases of serous or purulent infiltration of the membranes, or when false membranes had time to develop. The extent over which it is felt is not proportionate to that of the inflammation, for it may be felt all over the head, and yet the Meningitis be partial only; on the other hand, it may be exactly limited to one particular spot, and yet the inflammation be general. When partial, it does not always correspond to the exact seat of the inflammation, although when an individual complains of a fixed pain in a spot never varying, the probability is, that the meninges are inflamed at that point. The cephalalgia of Meningitis differs from that of continued fevers in its intensity, and in the fact that the patient does not wait, as in the latter, till asked whether he has any pain in his head, before speaking of it himself, and craving for relief. The character of the pain varies; it is described by some as a heavy weight pressing on their brain; by others, as consisting in violent shooting pains, either continuous or recurring at intervals. Sometimes, again, it is compared to the sensation of an iron band encircling the forehead, or of the head being squeezed in a vice. Vomiting is another well-marked symptom of acute simple Meningitis. It is purely sympathetic, and is unattended with epigastric pain or tenderness on pressure, or with nausea. It recurs very frequently for the first day or two of the disease, and may then cease; but in some cases it persists unto the end, either continuously, or with more or less prolonged intermission. The matters vomited are abundant in quantity, and are mixed with bile. The tongue is natural, generally moist, and occasionally covered with a white creamy fur. Constipation is the rule, and the discharges, when there are any, are dark and offensive.

The intellect is always affected from an early period. The temper is extremely irritable; there is marked somnolence or constant wakefulness, or the one may alternate with the other for several days. Delirium sets in early; it is generally of a wild, fierce character, the patient shouting and vociferating, and tossing himself about. In some cases, on the contrary, the first sign of intellectual disorder is obstinate mutism, the patient burying his head under the bed-clothes and refusing to answer questions. Sometimes the delirium reaches at once its maximum on its first occurrence, but otherwise it is slight at first and gradually becomes more and more marked. Sometimes, again, it is only nocturnal at the outset, and does not become diurnal until after a few days; and again, a patient who has been wildly delirious for a few days may recover his reason before death, although all his other symptoms grow worse.

From the beginning the patient staggers when walking, and his gait resembles that of a man under the influence of drink. When he has taken to his bed, he is extremely restless, and keeps constantly shifting his position. The muscles of his face and limbs, even in this stage, may begin to twitch involuntarily. When convulsions have opened the scene, as they frequently do in young children, they recur in frequent paroxysms one upon the other with scarcely any intermissions. The general sensibility is usually heightened in this stage, although it has sometimes been known to be normal. To sum up, the characteristics of the first stage are high fever, cephalalgia, an occasional sharp piercing cry, vomiting and constipation, general hyperæsthesia, sensorial and cutaneous, and fierce delirium. This stage may last only a day or two, but generally extends over a week, and sometimes over two weeks. It then merges into the second or transition period.

Stage Second.—The delirium becomes quieter; there is carphology, picking at imaginary flies in the air, or on the bed-clothes; and the patient's eyes become dim and lustreless: his pupils dilate, or they oscillate at first before they dilate, and become insensible to the influence of light. Vision is impaired; hearing gets dull. The patient complains less often of his headache, because he is less sensible; occasionally, however, he utters still a loud cry, he grinds his teeth, moving his jaw as if he were chewing, and rolls his head from side to side, boring his pillow with his occiput. Somnolence sets in, to be soon followed by a comatose condition. The pulse is less frequent, and occasionally intermits. The respiration is very irregular; at one time it is very rapid, and the next moment it is slow and suspirious, made up of a long, deep inspiration, followed by a slow and long-sustained expiration. The urine is retained, or there may be stillicidium after retention. The constipation persists; and the abdomen is apt to become retracted, sunken, and almost boat-shaped. The general hyperæsthesia is now replaced by hypæsthesia, which soon makes way for complete anæsthesia. The muscular twitchings increase; there is subsultus tendinum, and actual convulsions often set in. These may be general or partial, usually the latter, and they then attack different parts successively. The muscles most commonly affected are those of the eyeballs, producing strabismus, or rolling upward of the globes; the muscles of the face and lips; and lastly, of the extremities. The tongue itself may be the seat of convulsions. There may be rigidity in one or more limbs, and the head may be bent backwards, or be permanently inclined to one side. Spasm is apt, after a time, to alternate with paralysis, or the latter may be gradually or abruptly established. In some cases the paralysis may come and go; in others, a limb may be paralysed, whilst its homologue is the seat of violent convulsions. The characteristics of this second stage are generally prostration, convulsive movements, gradually developing coma and total paralysis, motorial and sensory, a thready pulse, and an irregular, suspirious respiration.

Stage Third.—The collapse is now complete, and the coma profound. The pupils are widely dilated, and are insensible to light, the eyes are half open, the face sunk and ghastly, the skin cold and clammy. The sphincters relax, the urine and fæces are passed involuntarily, and the pulse becomes frequent again, indeed more so than before; it is small, filiform, and uncountable; the breathing is stertorous, and the patient at last dies in a state of complete coma.

Acute Meningitis in old persons, Dr. Machlachlan asserts, seldom occurs suddenly, and rarely exhibits the acute symptoms of Meningitis which affects persons of middle age, or of a younger period of life. It commences insidiously, and without premonitory rigors; it may exist some days without the most experienced eye detecting it. The pulse is natural, the tongue clean, the bowels regular, and there is little or no vascular excitement, local or general. The temper is peevish and irritable, with more or less confusion of thought, inattention, and forgetfulness. The patient makes strange mistakes, takes possession of another's bed, uses the spittoon instead of the chamber-pot. When addressed, his replies are rational, but there is a peculiarity in his manner and expression of countenance, an apparent slowness of comprehension, and a vacancy of eye. The appetite meanwhile is normal, and there is no feverish reaction. Yet there is great restlessness, unsteady gait, a trembling hand when the patient lifts anything to his mouth. After from twelve to sixty-two hours there comes on slight feverish reaction, expressed by increase of temperature only, and not by flushing of face or increase of pulse. Next, wandering, low delirium, and incessant talking become frequent and characteristic symptoms. Maniacal excitement is uncommon: reverie generally passing into coma constitutes the rule. Headache is not a permanent symptom. The patient moans, but never complains. Unless the patient is pointedly asked about headache, there is never any allusion made to it, or to tinnitus aurium. Dr. Machlachlan takes especial notice of this absence of headache; for even in the most acute pus-forming or false-membrane-forming Meningitis, headache may be entirely absent from the beginning. The eyes are suffused, the pupils slightly contracted, or natural. Knitting of the eyebrows, intolerance of light, acuteness of hearing and vomiting, are comparatively rare in the aged. The only objective evidence of increased vascular action within the cranium may consist in a hotter scalp than natural, and suffusion and injection of the eyes. There is great thirst usually, the patient will drink freely when liquids are offered to him, but he will seldom ask for drink, whilst he is very apt to refuse all food. In the worst cases, nervous twitchings and convulsions are observed, and these symptoms may be induced when otherwise absent, by raising the patient in bed. When coma is present slight convulsions of the limbs may be present. The general aspect of the senile Meningitis resembles typhus fever rather than Meningitis. The dryness and brown colour of the tongue, the muttering delirium, excessive prostration, injection of conjunctivæ and heat of scalp, equally belong to either affection; but in Meningitis the livid hue of the surface, and the mulberry rash characteristic of typhus, are wanting, whilst the diffused heat of skin of the latter disease is absent, or limited to the forehead and scalp in Meningitis.

Varieties as to Seat.—Meningitis may be partial or general. When partial, it may be limited to a small portion only of one hemisphere, generally the anterior lobe, or to the whole of one hemisphere alone; in that case, stopping abruptly at the median fissure, or it may affect the convexity of both hemispheres, or be restricted to the base alone. In some cases it affects the base, and the convexity of the hemispheres simultaneously. Meningitis of the base alone is, in the immense majority of cases, of tubercular origin. Parent-Duchâtelet and Martinet regarded very profound coma, not preceded by delirium, as characteristic of this variety of the disease; but Andral has conclusively shown that they were mistaken. As regards the relative frequency of the partial and general form of Meningitis, Parent-Duchâtelet and Martinet say, that in ninety-one out of a hundred and seventeen cases, the inflammation affected the convexity of both hemispheres, and in twenty-six, one hemisphere alone.[1]

A very rare form of Meningitis is that which is confined to the membrane lining the interior of the lateral ventricles, a membrane which is so very fine and delicate, as to be indistinguishable when in a healthy condition, and the existence of which has even been denied by Kölliker. Andral relates five cases of this variety, in one of which the granular condition of the membrane pointed to its tubercular origin, a view which was supported by the presence of tubercles in one lung; whilst in another of these cases, the intra-ventricular effusion occurring in the course of general anasarca and ascites, was probably one of the results of albuminuria. The first case of the series, however, seems to be an instance of genuine inflammation of the membrane lining the lateral ventricles, which cavities were found after death to contain a sero-purulent fluid. The symptoms of this rare form of the disease do not differ from those of Meningitis in other parts. There is the same acute cephalalgia, generally referred to the supra-orbital region, followed by delirium and coma, and in some cases attended with violent agitation, convulsive movements, and tetaniform rigidity of the limbs.

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[1] Parent-Duchâtelet et Martinet, De l'Arachnitis, p. 94.

Cerebral complications are sometimes apt to arise in the course of an attack of acute articular rheumatism. Of these, an affection resembling Meningitis seems by far the most frequent. In his work on "Diseases of the Brain," Abercrombie relates a few instances of this variety of the affection in a chapter headed "A dangerous modification of Meningitis, which shows only increased vascularity." In France, where the affection seems to be more common than here, the subject has been very much discussed of late years and has given rise to a good many publications. So far back as 1835, however, Sir Thos. Watson had called attention to these head complications, which he ascribed to a disturbance of the cerebral circulation, arising from the cardiac disease, which so often supervenes in the course of acute articular rheumatism, and which he stated to denote an inflammatory condition of the brain or its membranes.

There is no doubt that cases of rheumatic fever have occurred in which, from the character of the symptoms, the existence of Meningitis has been diagnosed, whilst post-mortem examination disclosed no such affection. Several such cases are mentioned by Dr. Fuller, in his work on Rheumatism, pp. 303, 304.

The invasion of the disease is, as a rule, very sudden; mostly appearing in the middle of the night. There may or may not have been some premonition of the coming complication in the shape of a feeling of anxiety, of terror, of strange forebodings of evil, a fear of impending death, expressed by the patient (Bourdon and Vigla); or, for a few hours before the actual attack, there may have supervened sudden mistiness of vision, as in a case related by Trousseau, in his "Clinique Médicale." As a rule, the disease does not occur at the commencement of the rheumatic attack, but is always preceded by pain and swelling of one or more joints. The invasion itself is characterised by great increase of temperature, restlessness and jactitation, extreme loquacity, and wild delirium, soon followed by coma, with intervals of muscular twitchings or slight convulsions. In some cases, the delirium is remarkable for the obstinate taciturnity of the patient. The pulse suddenly becomes very frequent, small, and irregular. The respiration is sometimes hurried and sometimes slow.

In the majority of cases, the swelling of the joints goes down, the redness disappears, and the cerebral symptoms seem to be attributable to a real metastasis. In some cases, however, the articular disease persists undisturbed. As to the pain in the joints, it is no longer complained of; but this is no proof that it has ceased, it merely shows that the intellect is so affected that the patient is unconscious of pain, or at least no longer takes cognizance of it. One of the most distinctive features of this form of Meningitis is the absence of headache and the absence of vomiting; two symptoms which are so prominent in simple acute Meningitis. In the latter affection, the course of the disease is generally rapid, but its rapidity is much less than that of rheumatic Meningitis. The patient may die in a few hours; more often from two to five days. If he lives beyond that time, the probability is that he will escape; and this prognosis will be all the more certain if the swelling, pain, and redness of the joints again make their appearance. Recovery, however, is rare; but when it does occur, convalescence sets in rapidly. In some cases the affection terminates in insanity, but the rule is, that death takes place; according to Vigla,[1] thirty out of thirty-nine cases terminated fatally.

The causes of this peculiar complication of acute articular rheumatism are very obscure. It is said to occur more frequently in cold weather, in the cold months of the year; and there is no doubt that exposure to cold is often an exciting cause. Vigla's assertion that patients who perspire very profusely, and who show confluent sudamina, are more liable to the disease than others who perspire less and have few sudamina, can be easily explained by the greater liability of the former category of patients to catching cold. The disease has been attributed to the perturbing influence of certain methods of treatment. Repeated blood-letting has, by some, been regarded as the cause of the disease, on account of its weakening the system and causing a preponderance of the amount of fibrine over the other constituents of the blood (Beau and Briquet). Others, on the contrary, have ascribed it to the large doses of quinine which, in France particularly, are administered in acute rheumatism. The well-known influence, they say, of quinine in large doses, in producing tinnitus aurium, giddiness, and deafness, explains how its administration may, in acute rheumatism, render the brain liable to be affected by the rheumatic poison circulating in the blood. This influence of quinine in the causation of rheumatic Meningitis is far from being proved, and Trousseau denies it emphatically. This author cites a case of Dr. Beau in which the symptoms of Meningitis began to show themselves, after the commencement of the quinine treatment, when small doses only of the drug had been given, while it completely disappeared on continuing the treatment, and giving larger doses of quinine.

According to Trousseau, habitual excess in drinking is the chief cause predisposing to this affection, and also an hereditary tendency to neurotic affections of any kind, and chiefly the various forms of insanity. The post-mortem appearances found in such cases are generally a considerable injection of the membranes of the brain, and in some cases sub-arachnoid effusions of serosity are met with; in very rare ones, pus has been found over the hemispheres, as in three cases by Watson.[1] Trousseau regards the affection as a neurosis, and totally discards the opinion that it is really constituted by an inflammatory condition of the meninges, and it is now well known that in the majority of cases of acute rheumatism terminating by high temperature and head-symptoms nothing at all resembling Meningitis has been discovered upon post-mortem examination.

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[1] Actes de la Société Médicale des Hôpitaux de Paris, 1855, Troisième fascicule.

Meningitis occurs also in individuals suffering from tertiary syphilis; for just in the same way as nodes and gummy tumours form under the periosteum, in different parts accessible to view, similar deposits are found in the substance of the dura-mater. In some cases, the membrane is not inflamed in the vicinity of these growths, but in others the dura-mater is thickened, and adherent to the brain, while itself participates in its superficial layer, in the chronic inflammation. The symptoms indicating the presence of such deposits are intense and constant cephalalgia, with nocturnal exacerbations; in some cases with convulsions, obtuseness of the intellectual faculties, and sometimes paralysis. The previous history of the patient, the peculiar sallowness of his complexion, and the presence in many cases of periosteal nodes, either on the head itself or on the bones of the leg, sufficiently attest the nature of the case. In less patent cases, the history of the patient will often decide its character. According to Robin and Lebert, these deposits can be recognised to be of syphilitic origin by the characters which they invariably present, and the following is a description of their microscopical structure as given by Robin.[2]

First.—They consist of an amorphous, transparent, greyish, granular blastema.

Second.—This blastema is traversed in spots by lamellar fibres which lie deeply in it, and are often difficult to see, and which are accompanied with fusiform, fibro-plastic bodies, not numerous on the whole.

Third.—In the portions that are whitish, the amorphous blastema is scattered over with a certain number of fat granules, and some drops of oil.

Fourth.—The most abundant elements of the tissue are cytoblasts, which make up four-fifths of the whole. They are uniformly distributed in the amorphous blastema, and between the fibres of the lamellar tissue; they are separated from one another by a substance which scarcely equals their own width. From their number, and their mode of distribution, they make the blastema look of a remarkable uniform composition. Free nuclei are found in great abundance. A few cells are also met with, with pale, transparent, finely granular bodies; they are almost all spherical in shape, some are ovoid, a few angular: the nuclei have a distinct and generally dark contour. They have no nucleolus.

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[1] Principles and Practice of Physic, p. 302, vol. ii. Fourth edition.

[2] In Zambaco, Affections Nerveuses, Syphilitiques, p. 80.

Fifth.—A few rare embryo-plastic elements.

Sixth.—A few globules of pus.

Treatment consists in the administration of large doses of iodide of potassium, of mercurial baths, or, when pain and sleeplessness are constant, of calomel and opium at night, with salines and iodide of potassium during the day.

INFLAMMATION OF THE DURA-MATER is exceedingly rare, as an idiopathic affection, and generally comes under the cognizance of the surgeon as the result of a blow or a fall on the head. Abercrombie gives one case of spontaneous inflammation of the membrane which had come under his own observation; but even there, the disease had spread to the arachnoid. Inflammation of the dura-mater is apt to supervene in cases of chronic otorrhœa, an affection which frequently sets in after scarlatina, and sometimes also after measles and variola. There is at first merely a thick muco-purulent discharge from the ear, with some tenderness about the mastoid process, and this goes on for a long time, when suddenly the patient becomes dull and drowsy, complains of intense pain in the head, he then becomes delirious, and lastly passes into a state of coma. After death, the petrous portion of the temporal bone is found carious and softened, and the dura-mater overlying it is seen to be detached, inflamed, and generally bathed in pus.

The same series of phenomena may also occur in cases of chronic disease of the ethmoid bone; or inflammation may spread to the dura-mater from the membrane lining the cavity of the orbit in cases of wounds of the eyeball or fractures of the orbit.

The symptoms of the meningeal complication are well marked: there is considerable rigor recurring in paroxysms, followed by high fever, and so marked are the intermissions that the disease simulates an attack of ague. Headache is complained of, and the discharge of matter from the ear does not give ease. Vomiting is often present, together with hyperæsthesia of the retina. There may be slight convulsions, but these are never general, and never so marked and so violent as in cases of pure Meningitis. When there is inflammation of the sinuses in the head, which are formed by duplicatures of the dura-mater, secondary abscesses show themselves in distant and various parts of the body, in the joints of the big toe, the knee, the hip, the wrist, &c. The aguish aspect of the symptoms in such cases is extremely marked: there are strong rigors, followed by heat and clammy perspiration.

The treatment should have for its object to prevent, or at least to limit, the extension of the inflammation, by the application of leeches to the mastoid process, as soon as it is found to be tender, and subsequently by the use of blisters. The ear should be carefully syringed with warm water, and the pus allowed a free escape. When there can be no doubt that the meninges are attacked, the treatment to be recommended in Meningitis should at once be adopted. When secondary pyæmic abscesses have formed, the treatment should be of a stimulating character, combining the administration of wine and strong nourishing broths, with the use of bark and acids, quinine and iron, &c.

Instances of recovery have been recorded in cases when head symptoms have pointed to an extension of disease from the internal ear to the dura-mater, and Abercrombie has related a case of a young lady who, after the usual symptoms, lay for three or four days in a state of perfect coma. Her medical attendants thought her condition utterly hopeless, and continued to visit her as a matter of form. One day, however, they were agreeably surprised to find her sitting up and free from complaint; a copious discharge of matter had taken place from the ear with immediate relief, and she subsequently perfectly recovered.

PROGRESS, DURATION, AND TERMINATION OF ACUTE CEREBRAL MENINGITIS.—The progress of the disease is always continuous; there may be slight remissions, but never those intermissions which form such a remarkable feature of tubercular Meningitis, during which the apparent improvement is so great as to mislead the inexperienced into the belief that the patient is getting well.

The duration of the complaint is extremely variable. It has been known to be fatal in thirty-six hours (Rilliet and Barthez), but as a rule, death only follows at about the end of the first week, although it may take place at the end of the second, third, and even fourth week.

The termination of the disease is generally in death: very few cases recover, and only when active treatment has been employed at the very outset. It is doubtful whether any acute cases ever pass into the chronic form.

PATHOLOGICAL ANATOMY.—The post-mortem appearances necessarily vary according to the stage of the disease in which death has occurred. Thus, there may be only intense redness from increased vascularity of the membranes, which are also remarkably dry; or, if the disease has lasted a few days, there may be fluid effused on the free surface of the arachnoid, in the interior of its sac, and in the meshes of the pia-mater. The effusion may be serous, sero-purulent, or entirely purulent. It is rarely abundant enough to produce a distension and prominence of the anterior fontanelle, although a case is related by Abercrombie[1] in which "at an early period of the complaint, there was observed a remarkable prominence of the anterior fontanelle; in the second week this increased considerably; and in the third week it was elevated into a distinct circumscribed tumour, which was soft and fluctuating, and pressure upon it occasioned convulsion. It was opened by a small puncture, and discharged at first some purulent matter, afterwards bloody serum. After death the opening which had been made through the fontanelle was found to lead to a deposition of thick flocculent matter mixed with pus, between the dura-mater and arachnoid, and covering the surface of the brain to a considerable extent."

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[1] Abercrombie, Diseases of the Brain, p. 57.

When death takes place at the end of the first week, the pus is no longer liquid; its more fluid portion has been absorbed, and false membranes of a bright yellow colour are now found, which are not yet adherent to the arachnoid. The pus infiltrates the meshes of the pia-mater, and dips between the convolutions. The concrete pus is found in greatest abundance around the larger blood-vessels, and in the sulci between the convolutions of the upper and lateral portions of the brain. Sometimes, as in a case detailed by Rilliet,[1] one hemisphere may be seen covered with pus or false membranes, whilst the pia mater on the opposite side is merely infiltrated with serosity. The brain, in uncomplicated Meningitis, is not affected; it is generally of firm consistency, and sometimes even more firm than usual. If death has occurred at an early period of the complaint, between the second and fifth day for instance, the grey and white substances present scarcely any traces of injection. After that time, they may still be perfectly healthy, although in the majority of instances the grey substance is of a somewhat pinkish hue, whilst the cut surface of the white matter shows numerous red points. The whole mass of the brain is always firm, but the peripheral layer of the convolutions may be softened; and when stripped off, the pia-mater which adheres to it carries away some portions of it. The longer the duration of the disease, the greater the risk of this complication, although there may be exceptions to this rule. In very young children, according to Rilliet, the whole mass of the brain is sometimes soft throughout, and he ascribes this softening to œdema of the brain. The lateral ventricles may be found empty, or they may contain transparent serosity, or even pus, and, in rare cases, false membranes. According to Andral (Clinique Médicale, vol. v. p. 140), the presence of serosity should not be regarded as the result of a morbid process, unless the quantity amount to more than *one ounce* of fluid in each lateral ventricle.

ETIOLOGY.—A. Predisposing causes:—

First, Age.—According to Guersant (Dictionnaire de Médecine, art. Méningite) simple acute Meningitis may occur in the fœtus in utero, and is pretty frequent in new-born infants. After the age of two up to fourteen, it becomes rare, and yields in frequency to tubercular Meningitis, the two being then in the proportion of two of the former to twelve of the latter. After fourteen, it again increases in frequency, and particularly attacks individuals whose ages range from sixteen to forty-five.

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[1] Rilliet, De la Méningite franche chez les Enfants. Archives Générales de Médecine, 1846, vol. xii.

Second, Sex.—It is considerably more frequent in male adults than in women; according to Parent-Duchâtelet and Martinet, in the proportion of three males to one female.

Third.—Those trades or occupations which expose the individual to atmospheric changes seem to predispose to the disease. Thus masons, carpenters, soldiers, &c. seem to be more liable to it than other men.

Fourth.—The sanguine temperament, a short thick neck, hypertrophy of the heart, a very irritable temper, are said also to predispose to the disease, as well as the abuse of alcoholic liquors, excessive grief, and mental work.

B. *Exciting Causes.*—The most common are blows on the head, falls, and concussions, &c.; and more frequently exposure to a hot sun in tropical countries. The sudden disappearance of a chronic eruption about the scalp, *e. g.* chronic eczema or impetigo, has been known to be followed by acute Meningitis; but this cause is not so frequent as it has been held by some authors.

DIAGNOSIS.—It is extremely difficult to distinguish acute Meningitis from acute Cerebritis—as the two affections so frequently coexist; inflammation of the membranes having a tendency to spread to the substance of the brain, or the reverse obtaining. In simple Cerebritis, however, uncomplicated with Meningitis, the excitement is not so marked, the delirium is not of the same wild, fierce character, the pulse either does not rise above its natural standard, or falls below it, even down to sixty and fifty; it is, besides, irregular and varies considerably in its rate of frequency. There is also *tonic* rigidity of one or more limbs, followed by paralysis, which is permanent. In every case, however, the limitation of the inflammation can at best be merely suspected.

From *Delirium Tremens,* acute Meningitis may be distinguished by the absence of headache in the former affection, the peculiar trembling, the hallucinations and spectral illusions and fears of the patient, the character of the delirium, and the abundant, clammy perspiration. The previous history of the patient, besides, usually tells a long story of inebriation.

Typhoid Fever may be separated from acute Meningitis by the headache being less intense, by the frequency of the pulse, the presence of diarrhœa, the infrequency of vomiting, if at all present, the gurgling in the iliac fossa, and abdominal tenderness, the leaden tint of the countenance, and, after the fifth day of the disease, by the characteristic rose spots.

The points of distinction between the simple and the tubercular forms of Meningitis will be given when treating of the latter affection.

TREATMENT.—The treatment of acute Meningitis is only successful when employed very early in the disease, and carried out with energy. It resolves itself into three great remedial measures: first, blood-letting; second, hard purging; third, application of cold water or ice to the head.

Blood-letting.—The patient is to be bled in the sitting posture, from a large opening in a vein in the arm, and continued until syncope is induced. The bleeding is to be repeated as often as the symptoms require it, or to be followed by the application of leeches behind the ears and to the temples. Continental practitioners often prefer bleeding from the dorsal vein of the foot to opening a vein at the bend of the elbow. According to Guersant, in very irritable individuals who are very sensitive to pain, especially in very young children, the application of leeches to the head increases the restlessness and the headache, and he therefore recommends that the leeches should in such cases be applied round the anus or about the ankles.

When it is not considered advisable to repeat the bleeding, compression of the common carotids in the neck, as originally suggested by Dr. Blaud (of Beaucaire),[1] might be had recourse to, so as to cut off for a time the supply of blood to the head.

The application of leeches to the interior of the nostrils, or scarifying the membrane with a lancet, is a favourite practice with some of the German physicians, and must directly relieve the circulation in the head on account of the inosculation between the vessels which ramify in the pituitary membrane and those at the base of the brain.

Purgatives.—Active purging possesses considerable efficacy in the early stage of the disease, and materially aids bleeding in producing its full effects. Calomel, jalap, and scammony are the purgatives usually selected, but croton oil seems by far the best, from the ease with which it may be administered, even to children, and the certainty and rapidity of its action.

Mercury.—Apart from its purgative effects, it is a most valuable remedy in Meningitis. It should be administered in small and frequently repeated doses, so as to bring the system under its influence quickly; and this is best effected by combining, with its internal administration, the use of mercurial inunctions in the groin and axilla.

The application to the head of cold, in its various forms, should never be neglected. There is no remedy so effectual in lowering the heat of the head, calming the headache, and subduing the violence of the delirium. Simple compresses, kept wet with cold water, are the least useful form of using cold, as they are soon heated and become dry, and the alternation of heat and cold thus produced might be injurious by attracting more blood to the head. A bladder, containing pounded ice, or a mixture of common salt and ice, is an excellent mode of applying cold, because of the facility with which it adapts itself to the shape of the head. The most effectual method, however, is *irrigation*, i.e. allowing a small stream of water to run on the head from a small vessel placed above it. The effect of this is almost magical, but it should be used with great caution, particularly in children and aged persons, so that its sedative influence might not be too powerful. Previous to using any applications on the head the hair should be cut close or shaved, and this simple measure is sometimes attended with great relief to the patient. Simultaneously with these applications revulsives should also be employed at the opposite extremity of the body by wrapping up the calves of the legs in mustard poultices, or in blankets wrung out of hot water and sprinkled with turpentine.

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[1] Bibliothèque Médicale, vol. lxii. See also Valleix, Guide du Médecin practicien, vol. ii. p. 49.

When the disease has passed into the third stage, that of coma, the above treatment is no longer admissible. Blisters applied to the nape of the neck and behind the ears, are exceedingly useful; and if the coma be very profound, a cap of blistering ointment applied over the whole skull has been known to rouse the patient. Flying blisters, applied in rapid succession to the inner aspect of the thighs, the calves of the legs, or mustard poultices even, are then useful also to rouse the system. When collapse has set in, mercury and purgatives should, of course, be discontinued, and stimulants, ammonia, and bark had recourse to. The bladder should be frequently examined to prevent the accumulation of urine and its consequent evils.

Diet.—The diet, in the first stages, should be low; no solid food is to be given.

In the third stage, however, strong broths given in small quantities repeatedly and wine become essential.

The room in which the patient lies should be kept cool and dark, and well ventilated, and free from the slightest noise.

Sometimes after the acute stage of the disease has passed, and convalescence has begun, the delirium is apt to return.

The practitioner should carefully guard against the error of mistaking this condition for one of recrudescence; it is due to exhaustion, and, as such, requires a judicious stimulating plan of treatment. It may be known by the coldness and pallor of the surface, and the weak compressible state of the pulse.

When occurring in the course of acute rheumatism prophylactic measures should always be adopted, such as the avoidance of cold, clothing the patient in flannel, and carefully watching the condition of the joints: if there be retrocession of the swelling and redness and pain in the joints, while the patient becomes restless and loquacious, we must try and bring back the rheumatism to the joints by wrapping them up in mustard poultices or applying blisters. Opium and musk have been recommended, and Trousseau declares that he has cured three patients by the combined administration of these drugs, although he adds that two others got well without any active treatment having been employed. In any case, the alkaline treatment for rheumatism should be continued, and careful nursing and the administration of unstimulating food adopted.

CHRONIC MENINGITIS.—This is a very rare affection, and it is generally recognised after death only from the pathological appearances met with in the meninges—namely, thickening and opacity of the arachnoid, cellular adhesions between it and the pia-mater, development along the falx cerebri of the so-called glandulæ Pacchioni, which from their absence in infancy and youth are generally regarded as evidences of chronic meningeal irritation. In some cases, plates of osseous tissue have been found in the membranes; whilst during life, the symptoms presented by the patient did not point to any mischief in the head.

Troublesome headache, a disposition to somnolency, sometimes convulsive twitchings, and in children, vomiting, are regarded as symptoms which should excite suspicion of the existence of chronic Meningitis. Of that form of the affection—which is complicated with chronic inflammation of the superficial layer of the cortical substance of the brain, and is symptomatically characterised by lofty ideas, hallucinations, paroxysms of maniacal excitement and embarrassment of speech, followed by gradual general paralysis of motion, sensibility being scarcely affected, and terminating in idiotcy—we have nothing to say here, as the affection is always treated of in conjunction with insanity.

Although chronic Meningitis in middle age and in early life is of rare occurrence, it is not so rare in old age. Concerning it Machlachlan observes, "The disease may be of a chronic nature, *ab initio;* chronic in regard to the subdued and insidious nature of its symptoms, while at the same time it pursues a strictly chronic course, seemingly, now and then existing one or two years, and never following an acute attack of the disease. It is not an unfrequent result of albuminuria and repeated attacks of delirium tremens, or it follows gout and rheumatism. Chronic Meningitis in the aged is almost uniformly accompanied with great impairment of the mental faculties, frequently with thickness of speech, and paralytic weakness of the lower extremities, the gait being tottering and feeble. The energies of the system are reduced; all movements of the limbs are performed awkwardly, slowly, and with uncertainty. The appetite remains good; but digestion is slow, bowels are inactive, and the various excretions vitiated. Vertigo, singing in the ears, marked loss of memory, slowness of comprehension, periodical fits of passion, and occasional attacks of headache, with or without signs of high vascular excitement, are frequently observed. Sooner or later the invalid takes to his bed reluctantly. There he lies uncomplaining, vegetating, and gradually sinking, dying often in consequence of sloughs on the nates."

TREATMENT.—The diagnosis of this affection being so uncertain and obscure, it is clear that little is known regarding the mode of treating it. If the symptoms in the least show a tendency to assume the acute form, the application of leeches behind the ears, cupping the nape of the neck, and administration for a short time of small doses of a mild mercurial, would be called for. Otherwise the use of repeated blisters applied to the nape of the neck, and the internal administration of iodide of potassium, and occasional purgatives, seem to be the most rational treatment that can be employed. In the form occurring in old age, cold lotions to the head, and an occasional brisk purge followed up by an enema, are most advisable. The condition of the bladder must be frequently examined, and the catheter employed should retention arise; when, on the contrary, there is dribbling, a proper apparatus must be worn. Good nursing, cleanliness, the hydrostatic bed, are also essential. In the later stages of the disease, when the vital energies begin to part and fail, wine will be essential, but until then the treatment should be strictly antiphlogistic; the patient avoiding also all mental excitement, and, if not secluded, he should be kept tranquil both in body and mind.

Secondary Meningitis.—This affection sometimes shows itself in the course of one of the eruptive or exanthematous disorders, as typhoid fever, measles, scarlatina, and variola.

When Meningitis occurs in the course of an eruptive fever, some of its ordinary symptoms may either fail entirely, or be masked by those of the primary disease. Thus, the intense headache of acute primary Meningitis may be absent, or it may be slight only, and there may be no vomiting. The invasion of the superadded disease may, however, be recognised by a sudden slackening and irregularity of the pulse and respiration, by the pallor and anxious look of the face, and the extreme jactitation which ushers in the delirium.

TUBERCULAR MENINGITIS.

BY SAMUEL JONES GEE, M.D., F.R.C.P.

BY tuberculosis we mean that disease which is attended necessarily by the formation of miliary tubercle. Inflammation of the brain and its membranes, occurring as a consequence of the progress of tuberculosis, is called Tubercular Meningitis.

CAUSES.—This is not the place in which to set forth the knowledge which has been gained of late years respecting the etiology of tuberculosis in general. A few conditions which predispose to Tubercular Meningitis are all that need to be dwelt upon.

Be the case as it may with regard to the adult, I think that most physicians will agree that the tubercular disposition in the child is strongly hereditary; it will be found that a large majority of children attacked by Tubercular Meningitis come of families in which there are, or have been, sundry manifestations of a tendency to scrofulous or tubercular diseases. Yet in many cases no tendency of the kind can be discovered.

The influence of sex, season, or social position upon the occurrence of Tubercular Meningitis is quite insignificant.

Tubercular Meningitis may set in at any age. No doubt the disease is more common before puberty than afterwards: but it is, at present, impossible to procure numerical proof of the fact, and this on account of the comparative absence of children from the general hospitals into which adults are admitted.

Age.	Cases.	Age.	Cases.
Six weeks	1	16 to 20 years, inclusive	10
Two months	1	21 to 30 ,, ,,	12
2 to 4 years, inclusive	12	31 to 40 ,, ,,	5
5 to 7½ ,, ,,	11	41 to 50 ,, ,,	3
8 to 10 ,, ,,	13	51 to 60 ,, ,,	2
11 to 15 ,, ,,	9	68 years	1
Total .	47	Total .	33

However, there can be no doubt that Tubercular Meningitis is comparatively much more common under two years of age, and much less common after eleven years of age, than these figures would make out. At the Hospital for Sick Children the following cases of Tubercular Meningitis were examined *post mortem* between August 1862 and March 1871 inclusive:[1]—

Under.	1 yr.	2 yrs.	3	4	5	6	7	8	9	10	11	12
Males . . .		3	13	10	9	6	5	1		1		
Females .		6	5	9	3	2	2	4	2			

Being 48 males and 33 females. Seven cases of non-tubercular cerebro-spinal Meningitis were examined in the same space of time; whereof six were males, (at 6 months, 2, 3, 6, 8, 9, years of age) and one was a female of 20 months. No case of simple cerebral Meningitis, not traumatic, during that period. After puberty, both acute tuberculosis and Tubercular Meningitis are much more common in the female than in the male sex.

Symptoms.—The symptoms of Tubercular Meningitis sometimes break in suddenly upon what has seemed to be, so far, a state of perfect health; sometimes they are preceded by several weeks or months of indistinct poorliness: both groups of cases (seeing that in both the sure and certain symptoms of distinct disease are cerebral from the very first) may be classed together under the head of Primary Tubercular Meningitis. But, on the other hand, Tubercular Meningitis sometimes attacks a person who has already exhibited symptoms and signs of either acute general tuberculosis, or of local tuberculosis, acute or chronic: this may be called Secondary Tubercular Meningitis. Ordinarily its existence is apt to be overlooked, unless its special symptoms have been watched for; sometimes, indeed, the disease is wholly latent, and discovered *post mortem* only.

I. Primary Tubercular Meningitis:
  1. With premonitory symptoms.
  2. Without them.
II. Secondary Tubercular Meningitis, preceded by manifestations of
  1. General Tuberculosis.
  2. Local Tuberculosis: i. cerebral;[2] ii. thoracic; iii. abdominal.

The primary form of the disease with premonitory symptoms is the typical form.

PRIMARY TUBERCULAR MENINGITIS IN THE CHILD.

I. *Premonitory Symptoms.*

Their Character.—i. Loss of flesh is the most constant precursor; indeed, is almost constant; often the first, sometimes the only symptom: the child's face being very much spared, it is when the nurse comes to undress him that she finds the limbs to be losing their roundness, and the flesh to feel flabby. This loss of flesh mostly proceeds continuously; sometimes, however, the child will seem to pick up for a time and afterwards begin to waste again. ii. Loss of colour concurs: it is sometimes masked by a false colour in the cheeks, due to permanently dilated capillaries; it may be only now and then that this colour leaves the cheeks, and then the real paleness of the child is obvious. iii. The child is drowsy by day and restless at night; he is easily tired, and in the midst of play will lie down on the floor and fall asleep for a short time; at night he grinds his teeth, sleeps with his eyes half-open, starts, and cries out; he is sad, fretful, peevish, taciturn, and wants to be let alone: if for any reason a young child has been put to bed for a day or two, he will not take to his feet again. iv. Headache is frequently absent, rarely a prominent symptom, but mostly present to a certain degree: very young children will be observed to put their hands to their heads, and to toss their heads on their pillows; older children will say that their heads ache, and this especially after some exertion of mind or body; a child will go to school and come home complaining of his head: the headache is rarely severe, and mostly frontal: a strong light increases it. v. Feverishness is not always present; when it is present, it is noticed chiefly in the evening. I do not myself possess any thermometrical observations made during this period, nor do I know of any that have been published.[1] vi. Loss of appetite is common; vomiting uncommon; the bowels are confined, or relaxed, or quite regular in action.

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[1] Children under two years of age are not usually admitted.

[2] *I.e.* Tubercular tumours of the brain which have produced symptoms.

These symptoms are grouped in every manner possible; any of them may be absent, and how many soever be present, they do not justify more than a fear lest the condition should terminate in Tubercular Meningitis.[2]

They are sometimes due to progressive tuberculosis, but sometimes certainly to a catarrhal state or to simple nervous depression. The pyrexia is the most important symptom. When a child suffers, day after day, from a slight elevation of temperature above the normal, that elevation of temperature may be due to tuberculosis. Inasmuch as a catarrhal fever does not usually last longer than ten or twelve days; if the pyrexia do last longer than that time, and if the presence of continued fever or of a local inflammation can be excluded; the elevation of temperature is very probably due to tuberculosis. But in actual practice we are not often called upon to study the precursory symptoms of primary Tubercular Meningitis so closely. This is certain, that the most careful mother will often fail to observe any feverishness before the day of invasion, even though she have been disquieted for weeks or months by her child's steady loss of flesh and strength. The poorliness which follows acute specific or other diseases often passes uninterruptedly into the premonitory period of Tubercular Meningitis. Measles, so far as I have seen, is the most common antecedent, hooping-cough next, occasionally an attack of diarrhœa and vomiting, bad sore-throat, or hip disease.

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[1] The reader will bear in mind that *primary* Tubercular Meningitis only is under consideration.

[2] Refer to section on Diagnosis.

The Duration of this period is different in different cases. Sometimes, as mentioned before, there are no prodromata at all.[1] There are all grades between this extreme and the other in which a child loses flesh for four, six, or even more months before the invasion. One or two months may be regarded as the average; sometimes the prodromata last two or three weeks only. Again, in some cases, the precursory stage is interrupted by a temporary improvement in the health of the child.

2. *Invasion.*

By the invasion of Tubercular Meningitis I mean the period at which there occur such new symptoms as enable us to pass from the uncertainties of the prodromal stage, and to declare most positively that from this date, at all events, the child has sickened with hydrocephalus. The invasion symptoms are the first which make the friends of the child think him dangerously ill; the poor, as a rule, only now begin to seek advice.

Character of Symptoms.—(1) Vomiting is by far the most common special invasion symptom. In the majority of cases the frequent repetition of the vomiting makes it seem to be the most important symptom of the onset; but sometimes, although serving to mark the invasion, the sickness is subsidiary in urgency to the other symptoms. In the former group, the child may vomit incessantly with and without taking food; in the latter, the child may be sick only once or twice. (2) Convulsions are the next most frequent (though much less frequent) special symptom of the invasion; they, like the vomiting, may be repeated several times or not. Sometimes the attack is epileptiform, sudden, with complete unconsciousness, and yet without convulsive movements. It will give some notion of the comparative frequency of the different modes of invasion to mention that, out of twenty-five cases in which the invasion symptoms were carefully ascertained, vomiting without convulsions occurred in nineteen; vomiting followed by convulsions in one; convulsions without vomiting in two; several attacks of general rigidity, succeeded by vomiting on the third day, in one; in one an attack of temporary unconsciousness (epileptiform), followed by vomiting; and in the remaining case the invasion was marked by a notable and comparatively sudden increase in the severity of the premonitory symptoms (headache, drowsiness, loss of flesh), without vomiting or any motorial symptoms. The most important concomitant symptoms of the invasion are: the first occurrence or the increase of headache, or of the pyrexia, or of drowsiness: the co-existence of constipation as a rule with an occasional exception; there is often a change in the temper of the child, or some odd unreasonable behaviour; the character becomes morose, irascible, and obstinate.

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[1] It may afford some notion of the frequency with which the prodromata occur to mention that out of twenty-six cases of primary Tubercular Meningitis (the diagnosis having been confirmed by a post-mortem examination in all), there were only two in which premonitory symptoms had not been noticed. That sometimes the prodromal stage is absent, or so slight as to be unnoticed, even in children carefully looked after, I have no doubt.

Period of Occurrence.—From seven to twenty-one days elapse between the invasion and death; the average is fourteen days. "When the meningitis sets in suddenly without prodromata, its duration is from twenty to thirty days, rarely less, provided that no complication modify the course of the disease" (Rilliet, iii. 487)—a very necessary qualification.

3. *The Established Disease.*

It is well known that Dr. Robert Whytt first described the course of what he rightly called "the most frequent species of the hydrocephalus internus;" and so described it that his successors have not been able to add much that is useful to his description.[1] The disease depicted by Whytt is a clinical entity of the most definite kind; but when we come to use the phrase Tubercular Meningitis, and to frame a species of disease characterised by certain anatomical changes (namely, tuberculosis and inflammation of the meninges), we find that we have to do with a more comprehensive notion than that intended by Whytt. The disease of Whytt corresponds to primary Tubercular Meningitis, involving the base of the brain, and occurring in children. But Tubercular Meningitis may be secondary; and, when primary, may not involve the base of the brain, or may attack adults: to these forms of the disease Whytt's description ceases to apply. Nevertheless, the set of symptoms pointed out by Whytt remains the most common and best marked manifestation of Tubercular Meningitis during life, and may be deemed the typical form, and will therefore be first described.

The peculiar symptoms of Whytt's disease depend upon the fact that the meningitis affects the base of the brain; the tubercular nature of the meningitis plays no part in the production of these symptoms. For both tubercular and non-tubercular meningitis of the base are attended by precisely the same symptoms; and Tubercular Meningitis involving the convexity alone is not accompanied by the symptoms which are peculiar to meningitis of the base. It must therefore be clearly understood that the form of disease which will be first and most fully described is, in reality, dependent upon basilar meningitis; and that, in a given case, finding this condition to be present, we assume it to be tubercular also, because basilar meningitis (unaccompanied by spinal meningitis) always is tubercular.

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[1] Works of Robert Whytt, M.D. Edin. 1768.

A. Meningitis of the Base.

Whytt's first stage of dropsy of the brain includes the premonitory and invasion periods, previously described. The subsequent course of the disease he divides into two stages; the one being an earlier period during which the pulse is infrequent, and the other a later period of frequent pulse. This division of Whytt's is true to nature, but the pulse is a fallible criterion. In order to recognise that a patient is in one or other stage of the disease, the physician must look at the symptoms in a comprehensive spirit. There is an earlier period (the second of Whytt), in which the brain may be regarded as reacting under or against the disease: the symptoms are *sthenic;* headache, delirium, exalted sensibility, infrequency of the pulse, consciousness being retained. The disease goes on to produce destruction of the brain; the symptoms become such as are due to a steadily increasing *paresis* of the animal functions; stupor, insensibility, paralysis, frequency of the pulse—the third period of Whytt. To repeat: the observer must not narrow his view to the variations of a single symptom, or he will often fail to perceive the stages of Dr. Whytt, or even be inclined to reject them. In the second of the ensuing paragraphs I have noticed the variations of the pulse somewhat minutely, so as to show how little it is to be trusted as a certain sign of the stage of the disease.

(1) Digestive Organs—i. *Vomiting.*—As already mentioned, the vomiting of the invasion is ordinarily repeated several times. Sometimes the symptom is very urgent, occurs whether food has been taken or not; and this may be so every day for a week. When once the tendency to vomit has ceased for twenty-four hours, it does not ordinarily recur. ii. *Constipation.*—In the great majority of cases the bowels are constipated throughout the whole course of the disease; yet sometimes they are spontaneously relaxed throughout; more frequently (especially when hard purging has been part of the treatment at the beginning of the disease) they are relaxed without the use of drugs, towards the end. The constipation is not often obstinate; it is easy, for the most part, to procure an action of the bowels by common means. iii. *Retraction* of the walls of the belly is a symptom which will be met with at some time or other in nearly every case: sometimes present for a day or two only, and at no certain period; sometimes present all along. Acute diseases affecting the brain are the only acute diseases of children which, as a rule, cause great excavation of the belly. iv. *The Tongue* has no fixed character; it may be moist and clean. Aphthæ sometimes occur as death draws nigh.

(2) The Circulation.—i. *The Pulse* affords symptoms which have been much studied, and with good reason, for they are most important. *a.* Its frequency is diminished in the earlier and increased in the later part of the disease: this is the rule. The increase mostly sets in during the second week; sometimes much later (*e.g.* boy, of four years, eighteenth day = 84; 116, 128, 128, 168, on successive days, death on twenty-third), or earlier (*e.g.* girl, eight years, sixth day = 140). Sometimes the increase occurs only three or four days, sometimes eight or ten days, before death. Sometimes the increase takes place suddenly (*e.g.* boy, two years four months, seventh day = 72; eighth day = 164), sometimes gradually. After the pulse has been very frequent, it may again become comparatively infrequent (*e.g.* girl of one year and ten months, eighth day = 162; ninth = 144; tenth = 100; eleventh, day of death, = 180). This infrequency may persist and increase up to the day of death (*e.g.* girl, two years and six months, fifteenth day = 120; sixteenth = 140; seventeenth = 76; eighteenth = 70; *i.e.* day of death: cases of this kind die in a state of algidity unsurpassed in any other disease.[1] As to the absolute frequency, I have not known the pulse to fall below 64. At the invasion of the disease "the pulse is not much accelerated: 108, 112, 120 at most; sometimes it is even already slackened" (Rilliet, iii. 480). The frequency is at all times easily increased by movement of the body. *b.* Irregularity in the rhythm and inequality in the force of the pulse are two symptoms upon which great stress is laid in the diagnosis of Tubercular Meningitis. They coincide with the period of infrequency; not that an infrequent pulse is irregular at all times, but it will be found so, at least, now and then. During the period of increased frequency the irregularity is less easy to detect, and is probably really less common; yet a pulse of *e.g.* 170 will sometimes be found distinctly irregular. ii. The modifications in the *cutaneous circulation* are well displayed in the face. If habitually pale (as it sometimes is from beginning to end of the disease), a flush is easily produced by excitation of any kind—by moving the child, giving him to drink, brushing the hand over the cheek, and so on. Sometimes the flushing is spontaneous: limited to one cheek, or general as regards the head; constant, or alternating with pallor. The highly characteristic facies of acute brain disease[2] is chiefly effected by the congestion of the face. What is best seen in the face is observed to a less marked degree in the skin of the trunk and limbs; that is, the skin is injected at times. Spontaneous injection is often made evident by the unusually distinct white ring left by the pressure of the end of the stethoscope. As in the face, so in the skin of the trunk and limbs, injection, when not present, may be easily procured: draw the finger across the skin, and, in a few seconds, a red streak will appear along the track of pressure. This is what Trousseau has called the "tache cérébrale": for my own

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[1] Refer to paragraph on Heat of Body.

[2] See paragraph (8).

part I believe it to be a sign which does not possess any diagnostic value.

(3) The *Respiration* also yields symptoms deserving careful study. The frequency is increased, diminished, or at the natural rate. Increased frequency of pulse and respiration sometimes go together, but not always (*e.g.* boy of five years, P. = 190, R = 24; day before death). Irregularity and inequality are often very striking in later stages of this as well as of all other acute cerebral diseases of childhood. Sometimes the respiration is irregular only, sometimes unequal only, often both irregular and unequal: the child may remain for many seconds as if he had forgotten to breathe, then follow a few rapid respirations, then another pause, and so on. A careful examination will sometimes be necessary to detect irregular breathing. Sighing expiration is particularly common when children become half-insensible from Tubercular Meningitis.

(4) *Heat of Body.*—The first three or four days of the established disease are attended by what seems to be (judging by the hand) a distinct increase in the heat of the skin. This is followed by a period of low pyrexia, during which the temperature only occasionally exceeds 101°. I do not say that the temperature is not sometimes persistently higher, but I do not happen to possess notes of any cases in which it was so; whilst, on the other hand, for days together the temperature may vary between 96° and 98°. As death approaches (say for about the four days preceding death) cases have seemed to me to group themselves into three classes: in one, the state of moderate fever continues up to the very day of death; in another, the fever greatly increases before death; in a third, the body-heat falls below the standard of health. An example of each class will make this more clear:—

Day before death.		Third.		Second.		First.		Day of Death.	
Type 1.	Boy: 4 years.	99·5	102·0	99·0	101·0	100·5	101·5	99·0	101·5[1]
Type 2.	Boy: 2½ years.	97·0	99·3	99·2	101·0	100·0	103·0	104·4	107·25[2]
Type 3.	Girl: 2½ years.	97·8	96·6	96·2	93·0	82·8	82·1	80·5	79·4[2]

The minimum and maximum temperatures of each day are given.

The peculiar variety of ardent fever referred to the second type (the lipyria of Galen) is, perhaps, more common in Tubercular Meningitis than in any other disease.[3] "Heat of the viscera, as if from fire, but the external parts cold; the extremities—that is to say, the hands and feet—very cold" (Aretæus). A thermometer in the arm-pit will prevent our being misled by the coolness of the exposed parts. In the third type the algidity involves the viscera themselves; the temperature in the example adduced was taken by means of a thermometer kept permanently in the rectum; and, as the hyperpyrectic cases might deceive the hand applied to the limbs only, so might the state of algidity deceive the eye. The example chosen one hour before her death, when her temperature was 79·8°, her breath cold to the hand, and her pulse imperceptible at the wrist, still kept a little colour in her cheeks, and (except that her eyes were half-open) it would have been impossible for one merely looking at her to have said that she was not a tolerably healthy child calmly asleep. The pulse, as a rule, agrees with the temperature, rising in frequency as the temperature rises, and falling as it falls: no proportion is kept in the amount of the rise and fall. Excessive frequency of pulse may concur with a moderate elevation of temperature; or, reversely, the temperature may be high and the pulse infrequent (*e.g.* temperature 103°, pulse 72—a ratio really observed, and such as would serve to clench the diagnosis of acute disease of the brain). Heat of the head greater than of the rest of the body is a symptom far from always observed in the earlier stages of the disease, and still less frequently in the later.

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[1] One hour and a half before death.

[2] At very moment of death.

[3] See article on Scarlet Fever, vol. i. p. 342.

(5) Nervous System.—i. *Headache* concurs with the invasion, or, if present previously, is much increased then. The pain is mostly referred to the top of the frontal bone. The headache is tolerably constant, subject to paroxysmal exacerbations, and lasts until stupor sets in. The temporary increase of headache is sometimes made known by the moaning of the child; sometimes he cries out, "Oh! my head," or shrieks,[1] or holds his hands hard on his head; he greatly dislikes any disturbance, for that increases the headache. But it must not be supposed that headache of this severity is present even occasionally in all cases. ii. *Eye-symptoms* are very important. One pupil is often distinctly larger than the other: this state is present at some time or other in every instance of the disease, does not occur at any special period, and is not always constant; thus the inequality in size, present in the earlier part of this stage, not rarely disappears later on in the disease, in order, it may be, that the relation of size may be reversed for a day or two before death: or, sometimes, the variations are much more rapid. Dilatation and sluggish action of the pupils is the rule towards the end of the disease, but the absolute size of the pupils is of small value in diagnosis. Squint is present sooner or later in every case. Hemiopia may occur so early in the disease as to be discoverable (Trousseau, ii. 236, 237). Oscillation of the eyeballs, or of one eyeball (the other being fixed), is common in the later period.[2] iii. *Paralysis of the Face*, one eye opened less widely than the other, one nostril

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[1] I copy the following particulars relating to the "hydrocephalic cry" from Trousseau (Clin. Méd. 2me édit. vol. ii. 239):—"It is a single, violent cry, resembling the cry of a person suddenly exposed to great danger: the expression of the face is not that of suffering: any period of the disease may be attended by this cry, which may occur every hour, half-hour, or even every five minutes." Rilliet (iii. 503) does not consider this to be either a common or a special symptom—an opinion with which my own experience would lead me to coincide.

[2] For the ophthalmoscopic appearances refer to paragraph (6).

being rounder than the other, one corner of the mouth less acted upon by the muscles than the other, one side of the upper lip straightened—these are frequent concomitants of the later period. iv. *Paralysis of the Limbs.*—Quite towards the end of the disease we often observe one or more of the limbs to be unmoved, relaxed or feebly rigid, flexed or extended. v. *Convulsions and Rigidity.*—These have been already mentioned as occasional invasion symptoms. As terminal symptoms they are equally frequent, occurring on the day of death, the child perhaps dying immediately after a convulsion. Yet they are not to be trusted as a sign of impending death, or even of death likely to occur in a day or two. When convulsions have been invasive they do not necessarily recur. The following may be taken as an example of the state of a child in convulsions towards the end of the disease: he lies unconscious, whole skin injected, eyeballs drawn upwards and to one side, pupils large, one side of face more wrinkled than the other, teeth clenched, limbs rigidly extended—except the hands, the fingers of which are flexed—slight twitching movements of face and limbs, more marked on one side than the other, respiration laboured. As the child comes round the unconsciousness diminishes, pupils become smaller, he is left bathed in sweat. Sometimes the clonic movements are more marked. Sometimes the limbs are relaxed throughout, and the twitchings limited to the face and eyeballs. Permanent feeble spastic rigidity of one or more groups of muscles is common during the latter period; sometimes the rigidity is much stronger. Opisthotonos may be present during the last few days of life; it may be paroxysmal and last only a few minutes, or it may be continuous and last until death. In such cases I have not found any signs of inflammation about the cord or its membranes. Tremulousness of the limbs is very common. A shudder may be often observed to pass through the body from head to foot. Many other niceties of motorial symptoms might have been described: enough has been said to show the infinite variety present in Tubercular Meningitis. vi. *Sensation.*—Tenderness of the skin can be demonstrated to be present in some cases; it is often very obviously present in the scalp when a barber is employed to shave the head. Pains in the limbs are sometimes complained of early in the disease. Dislike of light is common at the same period. Blindness is difficult of recognition, because occurring late. All these symptoms are sometimes unilateral. vii. *Consciousness.*—The children soon become somnolent: they lie with their eyes shut or half-shut, reply to questions in a dry short way or by a nod; when raised up in bed they complain much, knit brows, throw head back, and slip down in the bed. They dislike disturbance extremely: will clench their teeth against food. As the somnolence increases, the children cease to speak, but they will put out their tongues when shaken and pertinaciously asked to do so; they then relapse willingly into their former soporose state. By degrees, or sometimes suddenly, the sopor becomes deeper; but not until near the very end, and not always even then, does the coma become so deep that the child will not withdraw his limbs (provided they are not paralysed or rigid) when pinched, and also give other signs of being discommoded. Inability to swallow accompanies the coma. Retention of urine is sometimes rather an early symptom. The consciousness may be perfect the day before death. The semi-coma may be continuous from the invasion to the end of the disease. Delirium is common, but is not a symptom of much value in diagnosis, prognosis, or treatment.

(6) *Physical Signs* of tuberculosis of the lungs are not often to be detected in cases which, by reason of their course, are arranged under the head of Primary Tubercular Meningitis. Yet occasionally, and that even when the foregoing poorliness has not been greater than usual, I have detected the signs of a cavity under one or other clavicle. Sonorous râles may be met with, sometimes a little mucous râle; and in exceptional cases, which are, nevertheless, cases of Tubercular Meningitis as opposed to acute tuberculosis, all the physical signs are present of that very fine capillary catarrh which, in children, is nearly always indicative of the co-existence of tubercle or pneumonia. This sign I have observed in Tubercular Meningitis, and in no other disease: namely, the chest heaves equally well on both sides, and yet over a very large part, or even the whole of one side, no respiratory sound is heard by the stethoscope. In a few hours this sign will have passed away. It is probably due to a slight pulmonary catarrh concurring with the respiratory unconsciousness of the brain disease. When the fontanelle is large, it is mostly distended; when small, the distension cannot be perceived.

Of late years the ophthalmoscope has taken an important place among the means for discovering Tubercular Meningitis. Actual tubercles may be occasionally seen in the ocular choroid during life; but this is not a common occurrence, and if the ophthalmoscope were useful in this way only, its use would be very small. Choroidal tubercle was discovered, in one case, six weeks before the invasion of Tubercular Meningitis: Fränkel, Virchow's Jahresbericht 1869, p. 621. Steffen found choroidal tubercle in four out of five cases of Tubercular Meningitis, and in three cases of the four during life: eod. loc. 622. It is by detecting changes in the vascularity of the retina that the ophthalmoscope renders real service. Whenever meningitis is basilar, we find congestion of the retinal vessels, and sometimes optic neuritis. It will be obvious that there is nothing peculiar to meningitis in these signs; and also, that to find a state of retina which is probably dependent upon intracranial causes must be a very important item in the diagnosis, when we are doubtful whether a convulsion or an attack of vomiting be due to meningitis or not.[1] Meningitis which affects the convexity of the brain, and spares the base, is unattended by any unnatural condition of the optic discs: of this fact I have seen two instances.

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[1] See especially Dr. Allbutt's papers "On Optic Neuritis as a symptom of disease of the brain and spinal cord." Med. Times and Gaz. 1868, vol. i. pp. 495 et seq.

(7) *Urine.*—In the case of a boy, aged four years, in whom it was necessary to employ the catheter, I had an opportunity of examining the urine. The following was the result:—

Day of Disease.	Water.	Urea.	Ch. Sod.	Phos. Acid.
19–20.	122 c. c.	5·07 grammes.	0·195 grammes.	0·432 grammes.
20–21.	122 c. c.	5·51 grammes.	absent.	9·367 grammes.
21–22, day before death.	171 c. c.	7·34 grammes.	absent.	0·583 grammes.
Mean of 3 days in healthy boy of same age.	431 c. c.	15·27 grammes.	3·062 grammes.	0·967 grammes.

Weight of body—case of meningitis, 19½ lb.
„ healthy child, . . 28 lb.

(8) *General Appearance: Summary.*—Although the individual symptoms which have been now described are grouped in almost every possible manner in the different actual examples of Tubercular Meningitis which we meet with, yet it may be well to recapitulate the chief matters in what has gone before, and so to arrange them as to form a sort of idea or type of primary Tubercular Meningitis in the child.

A boy of five years old, in whose parental antecedents there are signs of a tendency to tuberculosis, begins to feel poorly, to lose flesh, and to complain occasionally of his head; he is restless at night, and languid by day; his bowels are rather confined; he is subject to irregular feverish attacks. These symptoms last two months, and then, one day, the child vomits for the first time; during the next three or four days the vomiting is repeated several times; afterwards it ceases; at the same time the fever runs higher, the headache increases, the nights are noisy, the constipation is obstinate. About the time that the vomiting ceases, other symptoms pointing to cerebral disease appear; say, for example, on the sixth day after the first vomiting, he looks thin and pale; skin hot and dry; temperature 100·8° (evening); pulse 84, irregular; respiration 20, regular; tongue dry, red tip, light fur elsewhere; bowels not open; belly natural; converging strabismus of one eye; pupils of middle size, mobile, one larger than the other; he says he has headache, and points to his forehead as its seat; physical signs of chest are negative; tache cérébrale uncertain; the boy is quite rational, moves about in bed, sits up, answers questions, and the expression of his face is not peculiar: the diagnosis rests (and rests surely) on the previous history, the pulse, and the condition of the eyes. On the seventh day he is much the same; belly rather retracted; pulse 108, very irregular; respiration regular; temperature, 100·4° (morning) and 101° (evening). Eighth day: no marked change, rather lower; pulse 112, still irregular; temperatures, morning and evening, 100·2° and 100·6°: all the other symptoms remain unchanged. Ninth day: clearly much worse; consciousness failing; does not cry out; probably can still see; tache cérébrale easily produced; cheeks, habitually pale, easily flush; cannot sit up; no special expression in face; swallows well; pulse, 160, regular, weaker; eyes as before; arms very tremulous; temperatures, 101·2° and 103°. Tenth day: still worse, semi-stupor, cannot be made to speak: eyes only half-open; passes excreta under him; lies fidgeting and picking with tremulous hands; pulse, 168, regular; temperatures, 101·8° and 102·4°. Eleventh day: stupor greater; he occasionally moans; whole surface much injected, face and head greatly flushed, dusky; and as the child lies on his back, motionless, with his half-opened and prominent eyes, their corners filled with thick secretion, and the corneæ dusty and filmed, he has a look quite characteristic of hydrocephalus; pupils dilated; one eye fixed, and probably blind; swallows pretty well; belly greatly sunken; pulse, 180, regular, very weak; respiration, 15, irregular; temperatures, 101° and 103°. Twelfth day: stupor deeper still; moves limbs of one side feebly; those of the other side are somewhat rigid; swallows badly; pulse so frequent and feeble that it cannot be counted; feet cold; temperatures, 101·5° and 103·6°. The next morning he dies.

Duration.—The duration of primary Tubercular Meningitis with prodromata is from seven to three-and-twenty days. It has been already mentioned, on the authority of Rilliet, that when the prodromata are wanting the duration is from twenty to thirty days.

Remission in the gravity of certain symptoms is not uncommon in the acute cerebral diseases of children. The direct nervous symptoms are the most variable; the squint, the unequal or dilated pupils, the rigidities, and the somnolence. The variations in the last symptom are the most striking and deceptive, semi-stupor passing away so as to leave the intellect perfectly clear. But when once the physician has satisfied himself of the existence of meningitis, he should not let his diagnosis be easily shaken. As Rilliet observes, "the improvement does not show itself in all the symptoms;" the pulse remains irregular, it may be, the squint or inequality of pupils persists, and though the remission should last a day or two, the child will die as surely and as early as if all the symptoms had been continuous.

Termination.—Tubercular Meningitis, running the course which I have now described, has but one termination, and that is death. But it has been suspected, and with good reason, that recovery sometimes takes place in the earliest stage of the disease.[1] The probability of this opinion has been greatly increased by the result of ophthalmoscopic examinations. If a child become febrile, and convulsed, generally or partially; if it vomits, or complains of headache, and at the same time the optic discs be found congested, it is very likely that it has basilar meningitis. All these symptoms may pass quite away: but occasionally the patient is left more or less imbecile, or epileptic, or partially paralysed, or with progressive atrophy of the optic discs. In these cases, however, a recurrence of the disease is to be greatly dreaded.

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[1] See Dr. Allbutt's papers "On the diagnostic value of the ophthalmoscope in 'Tubercular Meningitis.'" Lancet 1869, vol. i. pp. 596 and 599.

B. MENINGITIS OF THE CONVEXITY.

No doubt Tubercular Meningitis usually affects the base of the brain; but it is equally certain that this is not always the case. And inasmuch as the most characteristic symptoms of ordinary Tubercular Meningitis are in fact the symptoms of basilar meningitis, it follows that when the meningitis is not basilar, it is not attended by those symptoms. That is to say, the vomiting of the invasion period, the constipation, the infrequent and irregular pulse, the unequal pupils, the ophthalmoscopic signs of disease, the strabismus and other local paralyses, are absent from meningitis which does not involve the base of the brain. A state in which general convulsions are either present or imminent, the intervals between the convulsions being occupied by tremblings and twitches of the limbs and face, turning of the thumbs in upon the palms, clenching of the fists, stiffness of the back, neck, and limbs,—in short, a convulsive state, which is constant (except perhaps quite at the close of the disease),—this is the prominent symptom of Tubercular Meningitis of the convexity. Add moderate pyrexia, and a pulse which is frequent and very variable in its frequency.[1] The clue to diagnosis is to be found in the acuteness of the disease, the convulsive state, and the constant pyrexia: the evidences of basilar meningitis and of cerebral abscess being wanting. Meningitis of the convexity runs a more rapid course than meningitis of the base: two weeks, one week, or even less, commonly see the fatal termination.

SECONDARY TUBERCULAR MENINGITIS IN THE CHILD.

As before explained, meningitis is called Secondary when its symptoms have been preceded by manifestations not to be doubted of tuberculosis elsewhere. It has also been mentioned that, with this condition, the onset of the meningitis is, as a rule, obscure; a fact which will not surprise the reader when he considers the nicety of the premonitory symptoms, and the slight prominence of the commoner invasion symptoms appearing in the midst of a state of tuberculosis already existing. A state of *acute* tuberculosis, we ought rather to say; for when the tubercular disease is of that chronicity which we sometimes see, lasting for years (insomuch that tuberculisation having probably ceased, the patient suffers from its permanent effects merely), the recurrence of actual tuberculisation is only somewhat less marked than its supervention upon a healthy state. The small number of cases then (with regard to children, very small) which belong to the latter class may be dismissed from further consideration, differing as they do from primary Tubercular Meningitis only in this, that they run more rapidly to death. On the other hand, in a case of acute tuberculosis the cerebral lesion may have reached the point of complete softening of the septum lucidum and fornix, and not have produced any symptoms of hydrocephalus which could be discovered even by the observer watching for them. Between these extremes with regard to curtailment of symptoms there are all possible grades.

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[1] I wish it to be understood that I do not speak of cases of cerebro-spinal meningitis.

Tubercular Meningitis, when secondary to cerebral tubercle, is attended by symptoms which are for the most part distinct enough. This, indeed, would almost follow from the law before laid down; for cerebral tubercle (that is to say, a tubercular tumour), which has caused symptoms whereby it has been recognised, must be so chronic that the onset of the meningitis is well marked. If, on the other hand, the symptoms of tumour have been so slight as to have been insufficient for its diagnosis, then the case is, for clinical purposes, primary Tubercular Meningitis. Masses of yellow tubercle are often found imbedded in the brains of children dead of hydrocephalus acutus, whereof neither the prodromata nor the symptoms had led us to suspect the presence of anything more than the constant accompaniments of the latter disease.

It is uncommon for meningitis to supervene upon chronic phthisis in children; when this does happen, the new disease has been, so far as I have seen, easy of discovery; the more easy, the more chronic the precedent disease. Contrariwise, meningitis which occurs in the course of acute tuberculosis of the pulmonary form is mostly latent; when not so, very rapid in inducing death.

When tubercular peritonitis which has been diagnosed is complicated by meningitis, the latter is of the curtailed kind, apt to be overlooked, being, as it were, rather the harbinger than the cause of death—affording another proof of the truth of that aphorism which may be here repeated under another form, that the more tuberculosis has involved the health at large, the more obscure are the signs of a sequential meningitis: its premonitory and invasive symptoms have been anticipated. I have not known meningitis to supervene upon tabes mesenterica of such gravity as to have been a disease by itself.

The recognition of the occurrence of meningitis in the course of acute tuberculosis, which has been previously known to exist, depends greatly upon the degree to which the brain becomes implicated. Cerebral symptoms may be well marked (though shortened in duration, reduced to a week or less) even when they have been preceded by such grave symptoms, independent of the brain, as have not permitted us to doubt the existence of acute tuberculosis. Acute tuberculosis, not primarily cerebral, assumes for the most part one of two forms, namely, the typhoid form, or the pulmonary form. I cannot do better than quote Rilliet's description of the *typhoid* form when it precedes meningitis :—"In rare cases the invasion symptoms are more acute and febrile than usual, the skin is somewhat hotter, the pulse somewhat more frequent. The child complains of his head and belly at the same time; he does not vomit, but his bowels are obstinately constipated; he does not shriek, nor sigh, nor grind his teeth. The symptoms last from six to twelve days; fever continued; tongue covered with a thick fur; belly somewhat swelled and tender. The child is drowsy, but easily roused; answers sensibly; no photophobia; pupils natural; pulse regular, equal, 120 or more; no spots or sudamina anywhere; facies not that of hydrocephalus. This state is followed by the second (established) stage of meningitis." Let me add, that I have remarked, in such cases, the tongue to be pointed, with a central white fur and red tip and edges, and the bowels to be spontaneously relaxed. The *pulmonary* form is more common. The child sickens with what seems to be a bad cold; rapid loss of flesh and strength; fever rather high. The catarrh continues, fever increases, dyspnœa and lividity ensue; the râles heard in the lungs become more and more abundant, fine, sharp, and metallic; the percussion note is high-pitched and hard, without losing in resonance. After two or three months from the beginning, symptoms of meningitis appear, more or less distinct, therein following the rule already several times laid down.

TUBERCULAR MENINGITIS IN THE ADULT.

It will be no small gain if, by treating separately of Tubercular Meningitis as it occurs in the adult, a single reader be put upon his guard against supposing that acute hydrocephalus is a disease peculiar to childhood. As a matter of fact Tubercular Meningitis in the adult is not often diagnosed: yet were every one to study acute tuberculosis in the child, and then to transfer the knowledge acquired to the investigation of the diseases of the full-grown, there would probably be no special difficulty in the recognition of Tubercular Meningitis at any age.

Meningitis, when intercurrent in the course of chronic phthisis, is characterised by more or fewer of the following symptoms:—Headache, complained of for the first time; or, if previously present, greatly increased in severity; mostly, but not always, very painful; frontal. Vomiting is an early symptom, occurring in almost every case: vomiting in uncomplicated pulmonary phthisis is uncommon, except when brought on by the violence of the cough. Convulsions, occasionally, mark the onset of the meningeal disease. Delirium, of a quiet talkative kind, ensues. Sometimes the patients become speechless; they make ineffectual efforts to answer a question, or they look steadily at the speaker for a few moments, and then, without any expression of face, turn the head away. Numbness, paralysis, rigidity, of a limb or of some other part, may be a very early indication of the affection of the brain. At the same time, the symptoms, so far as the chest disease is concerned, "abruptly improve or actually disappear." Then follow: comparative infrequency of the pulse, and irregularity both of pulse and respiration; squint; inequality of the pupils; the patient lies in a meditative, semi-unconscious state, then becomes more and more unconscious, while motorial symptoms, of any kind, ensue. The phthisis is not often advanced.

Primary Tubercular Meningitis is at least as common, in the adult, as secondary; and, like as in the child, the symptoms may be nearly wholly cerebral from the first, or may assume a typhoid character. In the latter case, the disease is rather acute tuberculosis than Tubercular Meningitis; the non-cerebral symptoms predominate, at least at first. In the other case, the symptoms do not differ from those previously described, as occurring in the child, either in their character or their order of appearance; headache, at the beginning, is mostly very severe, but is not always so. Vomiting, strabismus, diplopia, more or less loss of power over some part of the body, convulsions, numbness, dilated pupils, infrequency of the pulse, early delirium, the presence of any of these symptoms in an adult suffering from an acute illness, should suffice to put us on our guard: the physical examination of the chest does not often help the diagnosis. The duration of the disease is from eight to fifteen days.

DIAGNOSIS.—The diseases which are confounded with Tubercular Meningitis may be divided for practical purposes into two classes: the first comprehending those diseases which simulate the earlier, and the second those which simulate the later, periods of meningitis. And it so happens that the resembling diseases of the first class are not attended, and of the second class are attended, by organic lesions of the nervous centres, or their appendages: this, speaking generally. The difficulty is greatest in the diagnosis of the earlier, the premonitory and invasive, periods of Tubercular Meningitis from the diseases of the first class; and the reason of the difficulty is obvious, namely, that the premonitory symptoms of Tubercular Meningitis are common to many diseases; so that the physician, full of a just dread of tuberculosis, and not wishing to be confronted by meningitis unawares, is continually suspecting tuberculosis when it is not present. To have treated incipient hydrocephalus slightingly is a mistake which, once made, is not readily forgotten; the patient's friends, at any rate, will remember the failure in prognostics. Loss of flesh going on steadily is a symptom to which it is wise to give the worst possible meaning. Repeated vomiting, in the child or the adult, occurring as a new symptom during a state of good health, or after a period of poorliness, is worthy of all our attention. Very carefully do we examine a child who has had a convulsion, lest it should be the first warning of the existence of incurable disease.

The *First Class* of diseases includes:—1. Simple Exhaustion; 2. Derangement of the Alimentary Canal; 3. Typhoid Fever;

4. Scarlet Fever and Small-pox; 5. Hysteria; 6. Simple Convulsions; 7. Pleurisy and Pneumonia.

1. Simple exhaustion of the vital powers (Morton's nervous atrophy) sometimes occurs so acutely and reaches such a pitch as to be mistaken for tuberculosis, or, indeed, actually existing Tubercular Meningitis. The exhaustion may be *primary:* a child, without any obvious cause, or perhaps in consequence of a slight catarrhal state or change of diet, loses its appetite, and therewith its flesh; becomes pale, languid, and restless; there are no distinct dyspeptic symptoms; the nurse fancies that the child is feverish; the pupils are large, and do not act very readily; a convulsion, or a series of fits, may occur—no other of the symptoms of meningitis being present. Wary in our prognosis, we submit the child to the test of treatment. We order pounded meat, milk, wine, or brandy, the aromatic confection, or a mixture of muriatic acid, cinchona, and chloric æther; the next day we shall be able to prognosticate much less dubiously; possibly, in the end, we may really have warded off Tubercular Meningitis. The exhaustion which is *secondary* to acute diseases, and especially to the longest acute disease, typhoid fever, is sometimes so great as to be mistaken for established hydrocephalus. It is chiefly in the houses of the poor that we see children, wofully mismanaged during their illness, wasted past belief. The alimentary canal ceases to perform a single natural function; the disgust for food is complete, the children are not even thirsty; forced to swallow broth, or food which is called light by a foolish metaphor, vomiting ensues; the child is somnolent, yet extremely restless, lies rooting with his head in the pillow, tossing from side to side, waving his arms in the air, or constantly passing his hand over one side of his head; incessantly whining, occasionally screaming, and, if old enough, complaining, when asked, of severe pain in the head; the tongue rolls from side to side, the lips are dry and peeling; the eyesight becomes dim, the somnolence deepens into unconsciousness, and the child dies. *Post mortem* we find the marasmus has invaded the brain; it is small and very bloodless; the pia mater is watery. The sketch is from nature, and the possibility of mistake is more than a mere possibility. The diagnosis will depend upon the previous history of the case, upon the character of the symptoms, and the order in which they have been developed.

2. Derangement of the alimentary canal.—i. *Acute dyspepsia* causes symptoms which are almost identical with those of the earlier periods of Tubercular Meningitis. To take an example: a child of four years old, of a phthisical family, suddenly vomits several times, becomes feverish, complains of pain in his head, has no appetite, coughs a little; is very irritable, thick-looking, and heavy; greatly dislikes being touched; the bowels are confined. Occasional vomiting continues; in the course of a few days (measured by the thermometer, the pyrexia in such a case may last a week) the fever diminishes, the pulse becomes irregular and much less frequent. But, happily, at the same time the child begins to look brighter, and to sleep better. It

is important to bear in mind that during convalescence, even from so trivial a complaint as dyspepsia, the pulse of many children becomes actually infrequent and very irregular. To increase the difficulty, I have known one pupil to become larger than the other at the same time, and to remain so several days. A diagnosis off-hand is often impossible; there is no help for it but expectation—expectation of the active kind. The patient is visited more often, examined more minutely, and treated more carefully than if there were no doubt; at least he does not suffer, probably he derives benefit, from the uncertainty of the physician. ii. *Gastro-intestinal catarrh.*—A little child, who had lost appetite and flesh for several weeks past, has one day a fit, which lasts, say, a quarter of an hour; on the same day her bowels become loose; they remain so for a week, then she vomits several times. All this time there is more or less pyrexia; what heaviness there is, caused by the diarrhœa, tends to complicate the diagnosis. From one example the reader will learn all: diarrhœa, although no doubt an uncommon, is not an impossible, accompaniment of Tubercular Meningitis. When dentition coincides, the pain caused thereby is not always distinguishable from the headache of meningitis: nor must we attribute too much to dentition; I have known the canines to pierce the gum and Tubercular Meningitis to break out at the same time.

3. Typhoid fever resembles not so much Tubercular Meningitis as acute tuberculosis. But typhoid acute tuberculosis is sometimes immediately fatal by way of meningitis. With regard to children, the physician, when in doubt, is far more ready to suspect acute tuberculosis than typhoid fever. The difficulty is caused by the aberrant forms of typhoid fever which we meet with; cases with confined bowels, with an empty belly, with spots which are small, dusky, and hard to the feel, or even vesicular at the apex; cases which have the facies of acute tubercle; cases complicated with consolidation of one or other apex of the lung, and attended by universal mucous rhonchi. In the adult, however, typhoid fever is far more likely to be suspected than acute tuberculosis. The practitioner, if fully aware that acute tuberculosis does occur in the adult, will not fail of making the diagnosis as soon as it becomes possible. Diarrhœa may accompany acute tuberculosis. Acute tuberculosis, with or without meningitis, sometimes greatly resembles typhus fever.

4. Small-pox and scarlet fever, both of which invade by vomiting, may be attended at the same time by very severe cerebral symptoms.[1]

5. Tubercular Meningitis sometimes at first simulates the symptoms of ulcer of the stomach, or may assume a quasi-hysterical form.

6. Simple convulsions cannot *per se* be discriminated from those which are precursory of Tubercular Meningitis.

7. The vomiting of incipient pleurisy and pneumonia, if accompanied by convulsions, as may be the case, is apt to divert attention from the chest to the head. But acute tuberculosis tends to cause

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[1] See vol. i. pp. 336 and 436.

inflammation of all the serous membranes; and, as a matter of actual experience, I have heard the friction sound of pleurisy in cases of Tubercular Meningitis; so that, on the contrary, attention must not be diverted from the head to the chest. The râle of lobular pneumonia is less general than that which we hear in some cases of miliary tubercle of the lung. In the lobular pneumonia of children, chlorides are often present in the urine while the body-temperature is still high.

The *Second Class* of diseases which simulate Tubercular Meningitis includes:—1. Simple Meningitis; 2. Abscess of the Brain; 3. Thrombosis of the Sinuses of the Dura Mater; 4. Caries of the Atlo-Axoid Joint; 5. Arachnoid and Sub-arachnoid Hæmorrhages; 6. Intracranial Tumours; 7. Hypertrophy of the Brain; 8. Essential Brain Fever.

1. By simple meningitis is meant meningitis which occurs totally unconnected with tuberculosis. Simple meningitis of the convexity is not a common disease. The symptoms are the same as are described under the head of Tubercular Meningitis affecting the convexity of the brain;[1] but the course of simple meningitis is more rapid. Convulsions and pyrexia in children; headache, active delirium, and pyrexia in adults, are the symptoms present early in the disease. Unconsciousness ensues in a day or two; the duration of the illness does not often exceed a week.

Sporadic cerebro-spinal meningitis (the membranes of the base and ventricles of the brain, and the sub-arachnoid space of the spinal cord, being especially affected), totally unconnected with tubercle, has been, in my experience, comparatively common. Cerebro-spinal meningitis is frequently quite a chronic disease; when acute, it resembles Tubercular Meningitis of the base very strongly.[2]

2. Cerebral abscess occurs under several different circumstances, and differs accordingly in clinical details. The diagnosis of Tubercular Meningitis continually opens up the whole field of diseases of the brain; the ability to distinguish them greatly depends upon a full and minute knowledge of their history. The reader will refer to the special articles, and make the necessary comparisons for himself. i. Cerebral abscess of pyæmic origin may be dismissed at once as never complicating diagnosis. ii. Cerebral abscess due to suppuration of the pia mater, going on so far that numerous large collections of pus are protruded into the brain substance, which disappears by rapid atrophy, so far as I have seen, does not modify the ordinary course of simple meningitis. iii. Cerebral abscess (due to disease of bones of the skull or not) differs in its symptoms according as pyæmia is combined with it or not. Abscesses merely pushed into the brain, inasmuch as they are not commonly combined with disease of the sinuses, cause symptoms which are altogether those of an intracranial tumour.

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[1] Page 383.

[2] The symptoms are the same as those of epidemic cerebro-spinal meningitis, to the article upon which subject refer, vol. i. 2nd edit.

Abscesses which are separated from the diseased bone by a layer of brain tissue (often greatly altered), and which are therefore presumed to have originated in the very midst of the lobe affected, are for the most part attended by pyæmia, the symptoms being complicated accordingly. But pyæmia does not always accompany even these non-peripheral abscesses, and then the difficulty of diagnosis from Tubercular Meningitis is very great, especially if we bear these facts in mind: first, that it is not uncommon for otorrhœa to concur with Tubercular Meningitis; and next, that external otorrhœa, in cases of cerebral abscess due to disease of the pars petrosa, may not set in until a week before death, and may have been preceded by the gravest symptoms of that intracranial otorrhœa which destroys the patient. The pulse of cerebral abscess is more persistently infrequent than that of Tubercular Meningitis.

3. Thrombosis of the sinuses of the dura mater, when secondary to neighbouring inflammation, does not admit of diagnosis, unless there be present pyæmial symptoms and some obvious possible cause of disease of the sinuses; caries of the pars petrosa is by far the most common. Thrombosis secondary to debilitating causes may be suspected if signs of disease of the brain follow a profuse diarrhœa or hæmorrhage in a young child, but could hardly be distinguished from the simple exhaustion before described; while, on the other hand, I have known a decolorised softening thrombus to occupy the whole bore of the upper longitudinal sinus, to be attended by large sub-arachnoid hæmorrhages, and to have caused no symptoms during life.

4. Caries of the atlo-axoid joint may cause such brain symptoms as to lead to a suspicion of the possible existence of Tubercular Meningitis. It is well, therefore, in a doubtful case, to examine the cervical region carefully, so as to discover any thickening and swelling of the soft parts.

5. Arachnoid hæmorrhage, according to Legendre, may simulate Tubercular Meningitis.[1] This can be only in exceptional cases of the former rare disease.

6. An intracranial tumour at the base of the brain, of the soft sarcomatous kind, which approaches nearest to cancer in general appearance and in rapidity of growth, may cause symptoms which resemble those of Tubercular Meningitis so closely, that, for a week or two from the beginning of the disease, it may be impossible to arrive at a diagnosis. In the case of the tumour, the pyrexia ceases for several weeks before death, and the disease becomes of a more chronic character.

7. Local hypertrophy of the brain is sometimes attended by symptoms which, at first sight, are like those of Tubercular Meningitis. We discover afterwards that the hypertrophy is, comparatively, very chronic.

8. Every practitioner, from time to time, will come across an acute

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[1] Rilliet and Barthez, ii. 259.

febrile disease, accompanied by symptoms which seem to point unmistakably to some affection of the brain; there being every reason to exclude the notion of suppressed exanthemata or analogous disorders. After one or several weeks of coma, delirium, severe headache, or whatever may have been the prominent symptom, the patient recovers, and we are left quite unable to say what has been the matter with him. To go more into detail, I could not do otherwise than narrate a series of cases which would differ from each other in most important points, and have nothing in common excepting pyrexia and brain symptoms. There is, generally, something wanting which makes us suspect that we have not to do with Tubercular Meningitis. Brain fever is as good a name as any whereby to designate these different anomalies; cerebral congestion, which is more commonly used, involves an explanation which is probably often wrong, and certainly never proved to be right.

MORBID ANATOMY.—I shall describe the morbid appearances of Tubercular Meningitis in that order wherein they are brought under view during a post-mortem examination.

Separation of the calvaria is easily effected as a rule. Miliary tubercle of the most undoubted kind was once seen by me upon the inner surface of the *dura mater*. Slitting up the longitudinal sinus, a pale narrow clot is seen in the posterior half: sometimes the sinus is filled with fluid blood and loose coagula, sometimes with a large black shining thrombus. Removing the dura mater, the great *arachnoid* sac is found to be destitute of fluid; the membrane itself is dry, and, what is more, sticky to the finger passed over it. Scrape the surface gently with a scalpel and the sticky matter will be removed, minute in quantity, and puriform in appearance. Reddish serosity has been observed in the arachnoid sac by Senn and Becquerel; transparent or turbid serosity by Rilliet and Barthez. This serosity, was it observed before or after the brain had been removed? If after, the observations are quite valueless, unless indeed certain precautions were taken which probably were not. The ordinary unnatural state of the arachnoid may be looked upon as the sign of a feeble inflammation; similar stickiness is common in incipient pleurisy and peritonitis. Empis once found the arachnoid sac obliterated by old adhesions in a patient who had probably passed through an attack of acute tuberculosis long before. Still more to the point: in Tubercular Meningitis there is almost constantly present adhesion, more or less firm, of the opposed surfaces of the great longitudinal fissure, especially just above the corpus callosum.

The *pia mater* affords more unequivocal signs of disease. First, as to vascularity. Sometimes there is obvious hyperæmia of the whole convexity of the brain: it looks rosy; examined minutely, the fine vessels are seen to be injected everywhere; the body having lain on the back, the injection is nearly as well marked over the anterior as over the posterior lobes. More commonly, the capillaries are not much injected; what colour the surface has being derived from large veins full of blood. Sometimes capillaries and veins both are emptied of blood, so that the brain has a most striking appearance, exactly resembling in colour painters' putty. These differences depend for the most part upon the amount of pressure from within to which the surface of the brain has been subjected. Secondly, as to œdema. Excess of clear serosity is commonly met with in the meshes of the pia mater between the convolutions; sometimes the effusion is semi-opaque and lymph-like. Thirdly, as to the tubercles. Examine the membranes of the lateral regions of the brain, corresponding in position to the temporal fossæ, and almost certainly miliary tubercles will be seen; not that they are absent elsewhere, but they are most common at the spots indicated: they are common at the bottom of the great longitudinal fissure also. These tubercles are beneath the arachnoid, often adherent to its under surface; those exposed to pressure against the skull are more or less flattened. Alongside the branches of the middle cerebral artery it is common to find a firm, greyish, semi-transparent material, which is probably confluent tubercle. Minute opacities of the pia mater are sometimes seen in the same region, most numerous by far in the neighbourhood of the miliary tubercles, and possibly tubercular in nature, a sort of white "tubercular dust." Sometimes the tubercles are yellow at their centre, sometimes all of them are yellow throughout, remaining crude. The number of tubercles present may be very large; there may be none at all. Raising the membranes from the surface of the brain, small portions of the brain substance adhere to the membranes so as to be removed with them: not that this is always the case; the difference depends upon the degree which the softening of the cortex has reached. The amount of vascularity of the pia mater and the degree of cortical softening are not always in direct proportion. The *convolutions* of the brain are more or less flattened, the intervening sulci narrowed. Proceeding to slice the brain, we perceive that the colour of the cortex is increased in depth if the pia mater be hyperæmic, or diminished *vice versâ,* or remains natural. As a rule, the *centra ovalia* are anæmic, sometimes exceedingly so. Sometimes the texture of the whole brain is obviously softened. When we reach the *lateral ventricles,* they are found to be distended with fluid,—a colourless serosity, of low specific gravity, mostly clear, but becoming faintly turbid when agitated; slightly albuminous, containing chlorides and phosphates. The quantity of the fluid is from one to four ounces; sometimes more. In one case I found a drachm of fluid in the fifth ventricle, the septum lucidum being everywhere perfect. The foramen of Monro is dilated. The lining membrane (ependyma) of the ventricles is toughened, sometimes obviously opacified in places, especially in the sulcus between the corpus striatum and thalamus opticus. Viewing the surface of the lining membrane sideways, we see that it looks as if it had been sprinkled with the finest dust. It seems probable that this condition is mostly due to small heaps of cells, a commencing suppuration of the lining membrane;[1] sometimes the dusty look, in part at least, is due to a minutely wrinkled state of the ependyma, resulting from the stretching it has previously undergone. Occasionally we see larger granulations than those described, greyish elevations, something midway, in every respect, between the sandy specks and miliary tubercles. The whole ependyma down to the fourth ventricle may be thus granular, or this sanded appearance may be quite absent. The vessels of the ventricles, the choroid plexuses and veins of Galen, with their tributaries, are sometimes obviously full of blood, but more often not so, and sometimes almost empty, the plexuses being quite pale. Softening of the cerebral matter beneath the ependyma is almost always found; the septum lucidum and under surface of the fornix are reduced to a pulp; the corpus callosum, walls of the posterior cornua, and other parts are often similarly affected. The question naturally arises,—What are the causes of these lesions of the ventricles—the dropsy and the softening? We cannot suppose that the brain substance will soften by passive imbibition of fluid; were it possible, there would be no reason why softening should not occur in health. But is the fluid forced by its excess into the brain substance? This is not so, because the ventricles are sometimes nearly empty when their walls are thoroughly softened. Mechanical congestion might conceivably be the common cause of the dropsy and the softening; but, in the great mass of cases, it is impossible to discover any impediment to the return of blood from the ventricles. The blood may be made to flow from the veins of Galen back into the straight sinus with perfect ease; moreover, as stated above, the plexuses are often quite pale, and the veins nearly empty. It would be difficult to explain the effusion of serosity into the pia mater, and the cortical softening, by mechanical congestion. Inflammation will account for all the conditions. We have already seen that the ependyma presents an appearance common in inflammation of surfaces,—namely, proliferation of cells. The connective tissue which underlies the epithelial layer of the lining membrane is gradually lost in the neuroglia or interstitial non-nervous tissue of the brain. And hence, inflammation of the ependyma leads to effusion of serosity both in the ventricles and into the brain matter; the softening being rendered complete by the mal-nutrition which ensues in consequence of the more or less arrested capillary circulation. To proceed: the velum interpositum is natural, or œdematous, or thickened and somewhat opacified; studded, it may be, here and there with miliary tubercles. The condition of the *third ventricle* resembles that of the lateral ventricles: the distension is ordinarily less because of the resistance of the thalami optici: in front of these masses the dilatation sometimes goes so far as to expose the pia mater of the base: the commissura mollis is often more or less torn and speckled with capillary hæmorrhages. The *fourth ventricle* also is distended. I have several times examined the cerebro-spinal opening *in situ*, and have always found the membranes about it perfectly healthy. When the *spinal canal* is laid open before the calvaria has been removed, to puncture the sub-arachnoid space (internal arachnoid of Hilton) causes the escape of a certain quantity only of fluid, merely the excess in the spinal canal; when the calvaria has been removed before the spinal sub-arachnoid has been touched, the cerebral ventricles can be drained completely by opening the membranes of the cord. The spinal internal arachnoid is distended with fluid, especially around the cauda equina. I have never observed any other morbid condition within the spinal canal, but then it has been examined in a minority of cases. Removing the brain from the base of the skull, we occasionally find adhesions of the two surfaces of the great arachnoid about the circle of Willis. The membranes at the *base of the brain* are sometimes greatly injected, sometimes much less so. Miliary tubercles sometimes swarm in the Silvian fissures, interpeduncular space, round the crura cerebri, and on the top of the cerebellum: ordinarily they are not in very large numbers; occasionally there is only a tubercle here and there; still more rarely no unquestionable tubercles can be found. Sometimes the tubercle is of the crude yellow variety. Other parts of the membranes of the base than those mentioned sometimes present tubercles. Besides being tuberculised, the membranes (*i.e.* pia mater, sub-arachnoid) undergo those changes which have caused the name of meningitis to be given to this disease. The meshes of the pia mater are filled with serosity (clear or turbid), or with lymph-like material, or with puriform: sometimes the membrane seems to be merely thickened, toughened, and opacified. The inflammation of the pia mater is most marked in the interpeduncular space, but tends to spread forwards along the optic and olfactory nerves, sideways into the Silvian fissures, and backwards round the crura cerebri on the upper surface of the cerebellum, or right over the pons and as far back, it may be, as the medulla oblongata. There is no proportion between the amount of tuberculosis and of meningitis. Softening of the brain cortex is usual; the under surface of the anterior lobes, and the under surface of the cerebellum, are affected with especial frequency. Softening of the optic commissure and of the smaller nerves is mostly found. The sinuses at the base present nothing abnormal.

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[1] Löschner and Lambl: Aus dem Franz-Josef Kinderspitale, 1860; S. 82. Prague.

The foregoing description applies to the commoner form of Tubercular Meningitis, in which the membranes of the base of the brain are involved. In the less common form of the disease, limited to the convexity of the brain, the morbid changes correspond. The pia mater of the affected part is infiltrated with puriform lymph; the membranes of the base being spared: there is no excess of serosity in the ventricles.

It is obvious to the naked eye that the tubercles are everywhere in the closest connection with the blood-vessels, especially the small arteries. And, in fact, it is in the sheaths of the vessels that the tubercles are formed. Both by their position (being seated upon the vessels), and by their structure, recent miliary tubercles of the pia mater have the closest resemblance to the Malpighian follicles of the spleen: indeed the similarity is so great that when the same bodies, which are recognised elsewhere as being recent miliary tubercles, are found in the spleen, it is often impossible to distinguish them from the Malpighian follicles. There can be no reason for doubting that the meningeal tubercles are not of the same nature as miliary tubercles elsewhere. It is true that the lymphatic character of the vascular sheaths is very well marked in the brain, but the vessels of many other parts are similarly constructed, to say nothing of the lymphatic tissue which exists beneath the serous and mucous membranes, and in other places. According to Dr. Bastian, the new formation in the sheaths of the vessels of the velum interpositum is sometimes so abundant as to cause the vessels to be obstructed.

Accidental Lesions.—1. Masses of yellow tubercle are often met with in examining the brains of children dead of Tubercular Meningitis. Sometimes the tubercle is softened. 2. Capillary hæmorrhage coincides with the softening of the cerebral matter, when the softening has reached a certain point. Most frequently seen in the soft commissure, hæmorrhage sometimes occurs in other parts, the brain proper, the pons Varolii, &c., leading to utter disorganization of the tissue. 3. I have seen miliary tuberculosis of the brain substance carried almost as far as can be conceived possible—a whole hemisphere of the cerebrum so much softened that it was easy to wash all the brain matter away; which done, there remained a close network of injected and dilated capillaries studded everywhere with miliary tubercles. The meningitis in this case ran a very rapid course. 4. Meningeal apoplexy, and a decolorised thrombus of the superior longitudinal sinus, I observed in one case of tuberculosis of the meninges, unattended with obvious inflammation of them.

Other Organs.—Tuberculosis of the lungs, liver, spleen, lymphatic glands, kidneys, and ocular choroid, concurs with the brain disease. The tubercle is mostly miliary, sometimes yellow, crude, or softened. A girl of four[1] died on the tenth day of Tubercular Meningitis without prodromata; *post mortem* we found numerous miliary tubercles in the pia mater, great ventricular effusion, very little lymph at the base of the brain, and absolutely no tubercle in any other part of the body (which was carefully examined), excepting a small mass of cheesy material in each lung. Tubercular ulcers of the intestines are often present: also intussusceptions, easily reduced. The children will have frequently preserved a large amount of subcutaneous fat.

PROGNOSIS AND TREATMENT.—The prognosis must always be unfavourable; and when the disease has passed beyond the invasion period and has become established, recovery may be deemed hopeless. In the latter case, if we reckon twenty-one days from the invasion symptoms we shall probably cover the fatal termination. When the disease does not pass beyond what seemed to be the invasion symptoms of Tubercular Meningitis, the patient's recovery is not always complete; this has been already dwelt upon: moreover, a second attack sometimes ensues.

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[1] Under the care of Dr. West in the Children's Hospital.

What, then, is to be done by way of prevention of the disease? The prophylactics and ordinary hygienics are the same—animal food, change of air, warmth to the surface, moderate exercise; to which may be added cod-liver oil and cinchona. The bad prognosis of confirmed Tubercular Meningitis does not belong to acute tuberculosis. No doubt many persons recover from acute tuberculosis: knowing this, any patient suffering from what is possibly acute tubercle should be treated very carefully, so as, if possible, to stop the disease and prevent affection of the brain. If the patient be seen during the invasion period, he should be put into a dark and quiet room; be carefully and regularly fed; symptoms should be treated; constipation relieved; a convulsive state diminished by full doses of bromide of potassium. There can be no doubt concerning the powerful depressing influence exerted by continuous cold applied to the head: this means should be therefore employed if the disease be seen in its earliest stage. Cod-liver oil may be tried at the same time. Later on there is not much that can be done. Sufficient liquid food should be given to the patient, by means of a syringe placed between the teeth, if need be. Leeches, active purging, blistering, and such-like measures, will rather hasten the advance of death. It is best not to shave the head unless it be necessary to apply cold. If the corneæ begin to ulcerate, it is as well to keep the eyelids closed by means of a little sticking plaster.

CHRONIC HYDROCEPHALUS.

By J. Spence Ramskill, M.D.

This disease is a real dropsy occurring within the cranial cavity. The fluid may be collected in the sac of the arachnoid or in the ventricles of the brain, beneath the arachnoid membrane. The affection may be congenital or acquired. When congenital, it is generally, but not invariably, due to an arrest of development of the cerebral mass, although even in such cases the dropsy has been regarded by Rokitansky and Vrolik, whose opinion is quoted and endorsed by Dr. West,[1] as not a mere passive dropsy, but as the result of a slow kind of inflammation of the arachnoid, especially of that lining the ventricles, which may have existed during life. Such inflammation may also attack the child after its birth, and "each year," says Dr. West, "leads me to estimate more highly the share of inflammation of the lining of the ventricles in the production of Chronic Hydrocephalus. Acquired hydrocephalus begins to show itself about the period of the first dentition." According to Dr. West (p. 124), out of 54 cases, 18 of which came under his own observation, some indications of the disease were observed in 50 before the child was six months old; in 14 the symptoms existed from birth, and in 21 more they appeared before the completion of the third month. In some rare cases, the disease attacks children seven, eight, or nine years old, who until then had seemed to be free from all cerebral complaint. In some extremely rare cases, this affection has been known to attack persons of advanced life. Sir Thos. Watson[2] cites several instances of the kind, one of which occurred under his own observation. A young and distinguished lawyer of his acquaintance had one or two attacks of rather sudden loss of consciousness, while engaged in the Court of Chancery: by degrees he became dull, stupid, forgetful, and at length insensible. In this condition he died. A large quantity of serous fluid was found distending the ventricles of the brain. No other alteration could be detected. A case of Dr. Baillie's is quoted by the same author, the patient being a man fifty years old. The celebrated Dean Swift died of this complaint at the age of seventy-eight, three years after the commencement of the disease.[1]

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[1] Diseases of Children, p. 121.

[2] Practice of Physic, 4th edit. p. 464.

Gölis also mentions three instances in which this affection began in advanced life: two of the patients were about seventy years of age; the third, who was a physician at Vienna, likewise died in the decline of life, having suffered under the disorder for ten years. When hydrocephalus shows itself some time after the birth, it is generally accounted for by the presence of a tumour (cancer, tubercles, or cysts). The dropsy in such cases is produced, as was pointed out by Dr. Whytt long ago, by the same mechanism as ascites in cases of schirrhus of the liver, of the spleen, or of the pancreas. Any deposit compressing the veins of Galen, which bring back the blood from the ventricles of the brain, is sure to lead to accumulation of serosity within those ventricles.

Dropsical effusion within the sac of the arachnoid is sometimes the result of a former hæmorrhage into that cavity, as pointed out by Legendre, and supported by Rilliet and Barthez. In some very rare cases, Chronic Hydrocephalus seems to be a result of the acute disease. Two cases of this kind are recorded by Rilliet, one in his work on "Diseases of Children," p. 162, and the other in the "Archives générales de Médecine," Dec. 1847. Dr. West also relates a case in which the first link in the chain of morbid processes seems to have been an injury to the head; the child, when five months old, having fallen out of the arms of the person who was nursing her, and on the same day she had a fit, and remained stupid and senseless for hours.

Anatomical characters.—1. *Ventricular Hydrocephalus.*—The quantity of fluid varies from a few ounces to a few pounds. In a case mentioned by Trousseau,[2] the head measured a *mètre* (39·3 inches) in circumference, and about thirty pounds of fluid were found in the ventricles. The same author cites another case from Franck, in which the fluid amounted to fifty pounds.

As a necessary consequence of the accumulation of fluid, the ventricular cavities are considerably enlarged, the openings through which they communicate with one another are considerably dilated, although in some instances, from the pouring out of lymph, these apertures may get closed, and the fluid may therefore accumulate in one part more than another, producing an unsymmetrical enlargement of the head. Thus, Vrolik[3] has related the case of a young man who died from Chronic Hydrocephalus at the age of twenty, and in whom a false membrane had occluded the foramen of Munro through which the two lateral ventricles communicate.

The walls of the dilated ventricles may be of normal consistence, or even of greater consistence than normal. Rilliet and Barthez state that they have been able, in some cases, to dissect the condensed mass into several layers. In other cases the walls feel softer, and œdematous for some little distance. The brain mass above the ventricles becomes thinned and unfolds itself. The convolutions are flattened out, and the sulci between them disappear. The cerebral substance looks pale and anæmic. In some cases it happens that the commissures of the brain yield, and that the whole, or a portion of the fluid which it contains, escapes into the cavity of the cranium. This appears to have taken place in the well-known case of Cardinal, whose skull contained seven or eight pints of fluid, while "the brain lay at its base with its hemispheres opened outwards like the leaves of a book."[1] When the accumulation of fluid has resulted from inflammation of the membrane lining the interior of the ventricles, that membrane is found thickened and rough, and in some cases in a granular condition.

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[1] Practice of Physic, 4th edit. p. 464.

[2] Trousseau, Clinique Médicale, 2e édit. p. 247.

[3] Traité sur l'Hydrocéphalie interne. Amsterdam, 1839.

2. *Intra-arachnoid Hydrocephalus.*—When the result of hæmorrhage into the arachnoid sac, the fluid is found more or less yellowish in colour, and may be even more or less mixed up with thin, serous blood. When it has been poured out to fill up the vacuum in the skull due to defective development of the brain, it is perfectly limpid and clear. The sinuses of the dura mater in this, as in the preceding form of hydrocephalus, are either empty, or are found to contain blood, both liquid and coagulated. The fluid of hydrocephalus, when tested by heat and nitric acid, is found to contain albumen; chloride of sodium, soda, and traces of salts of lime and potash have also been found in it. Urea was detected by Dr. Bostock in his examination of the fluid found in Cardinal's head.

Condition of the bones in Chronic Hydrocephalus.—They are generally found to be considerably thinned, and transparent; if the union of the sutures has been completed, the bones are found to be less firmly united than usual, with less dovetailing; and there are numerous ossa triquetra found in the lines of the sutures. In some cases, the bones have been found of normal thickness, and in rarer ones they have been of greater thickness than normal (Rilliet and Barthez), hard, compact, and resisting.

Symptoms.—When the disease is congenital, signs of cerebral disturbance manifest themselves very soon after birth. There may be either strabismus and rolling of the eyes alone, soon followed by gradual enlargement of the head, or convulsions recurring pretty frequently may set in.

According to Dr. West,[2] "enlargement of the head is by no means invariably the first indication of Chronic Hydrocephalus. In twelve out of forty-five cases, fits, returning frequently, had existed for some weeks before the head was observed to increase in size; in six, the enlargement of the head succeeded to an attack resembling acute hydrocephalus; and in four other instances it had been preceded by some well-marked indication of cerebral disturbance. In the remaining twenty-three cases no distinct cerebral symptoms preceded the enlargement of the head." Failure of nutrition is almost invariably present, although Rilliet and Barthez assert that "the nutritive functions are as a rule well performed in hydrocephalic children, unless they be in an advanced stage of tubercular cachexia, or chronic intestinal catarrh. Except such cases, the children are plump and well nourished, and even have sometimes an abundance of fat which is certainly morbid."[1] The cases, however, in which nutrition is unaffected, form the exception, not the rule. The child sucks well, voraciously even, and yet does not grow; he may even waste. His bowels are generally constipated, and his motions are unhealthy. The gradually increasing head soon attracts notice, and the peculiar physiognomy and aspect of a hydrocephalic child soon develop themselves. The fontanelles enlarge, and the anterior one is seen often to pulsate, and grow tense and prominent; and at such times there is heat of the head, and the child is more restless than usual. The sutures of the head widen, and the head by degrees assumes a globular shape. The forehead is round and prominent, the orbital plates of the frontal bone gradually become slanting, and the eyeballs become half hidden under the lower eyelid, so that the cornea cannot be seen until this is depressed. The parietal bones being pushed outwards and their edges being last to ossify, there is a considerable increase of the sagittal suture, whilst the occiput is driven downwards and backwards, in some cases to such a degree as to be almost horizontal. On applying the hand over the opened sutures and fontanelles, a distinct sensation of fluctuation is perceptible. The hair grows very scantily on the head, on which very large distended veins are seen to ramify. The face is small, and contrasts remarkably with the large size of the head; and looks triangular, with the apex of the triangle at the chin. The child's expression is dull and stupid, and he has a very aged look: he cannot sit up, or hold up his head, but lies down constantly. As the fluid continues to accumulate, and the disease progresses, the sight becomes impaired, and is completely lost after a time; the eyes are bright and shining, but restless and oscillating. Hearing is as a rule preserved much longer, but is lost at the close of the disease. Paralysis often sets in; contractions and rigidity of the limbs and trunk are not very rare, according to Rilliet and Barthez, particularly in very young children (p. 160). Occasional attacks of laryngismus stridulus are not infrequent, and they may even come on before there is much enlargement of the head (West). That form of Chronic Hydrocephalus which results from the transformation of a cyst, the result of hæmorrhage into the arachnoid sac, may be recognised, according to Legendre, "by its being never congenital; by generally beginning about the tenth month, that is to say, about the time when the teeth begin to appear. The head, indeed, enlarges gradually, but does not acquire so large a size as in internal hydrocephalus; while, lastly, it is always preceded

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[1] Bright's Reports, vol. i. part i. p. 433.

[2] Diseases of Children, p. 121, 5th edit.

[1] Mal. des Enfants, p. 161.

by convulsions, or by some other form of active cerebral disturbance, which marks the date of the occurrence of hæmorrhage."[1] At best, however, the diagnosis can be but hypothetical. When hydrocephalus becomes developed after the sutures are united, the bones, being subjected to pressure, become thin, and in some cases the sutures have been known to give way. Such cases are spoken of by Rilliet and Barthez, who also quote from the *London Medical Journal* (for 1790, p. 56) the case of a child who at the age of nine years, and eleven months before his death, became affected with chronic cerebral symptoms. Nine months and a half after the first manifestation of the disease, the sutures of the cranial bones, chiefly the coronal, began to open. At the time of his death the distance between the edges of the coronal suture measured half an inch, and at the spot where the lambdoidal joins the sagittal suture there was a marked opening, so that the occipital bone was completely free.[2] As a rule, however, when hydrocephalus begins after the sutures are united, the head does not enlarge considerably, although it may do so in some rare instances, as in a case mentioned by Rilliet and Barthez (p. 165), of a child nine years old, who from the age of eight exhibited the symptoms of hydrocephalus, and whose head became enormously enlarged in spite of the ossification of the fontanelles. The size of the head in Chronic Hydrocephalus varies considerably; it has been known to measure two and even three feet in circumference. In the Museum of the Faculty of Medicine of Paris there is a hydrocephalic skull which measures 39 inches round. The shape of the head is generally globular and flat at the top, but in some rare cases it is conical, shaped like a sugar-loaf.

The termination of the complaint is generally in death, which occurs either from some intercurrent affection, hydrocephalic children being always weakly and unable to resist disease, or from an attack of laryngismus stridulus, or from convulsions due to passing congestion of the meninges, or lastly from gradual exhaustion, from positive asthenia. The disease extends at least one or two years, but it may last from four to ten years. Cases have even been recorded of individuals living to an advanced age who had been hydrocephalic from infancy. Thus Dr. Bright's patient, Thomas Cardinal, lived to nearly thirty. Franck, cited by Trousseau,[3] speaks of two individuals, the one aged seventy-two, and the other seventy-eight, who had been hydrocephalic from infancy. Strictly speaking there is no cure of the complaint, but merely an arrest of its progress. Fluid may be no longer poured out, but that which has been already effused is not absorbed. The sutures and fontanelles ossify and close, and a good many ossa wormiana are then found along the lines of union: these are like nuclei for the formation of bony matter. In some instances it has been said that a real cure takes place; that there is increased activity of the nutrition of the brain, producing hypertrophy of that organ, the fluid being absorbed and new matter deposited in its stead (Otto).[1] Such cases, however, must be quite exceptional, and the rule is that the fluid is unabsorbed and remains in the cranial cavities. The patient's intellect and senses are not perfect it is true, but are still sufficient to enable him to perform the ordinary duties of life, although he is apt to be fretful and irritable, and somewhat childish in his ways.

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[1] Legendre, Recherches Anatomo-pathologiques, p. 135.

[2] Sir Thos. Watson (p. 464, 5th edit.) also cites two similar cases, one from Dr. Baillie—the patient was a boy, seven years old; and the other from Dr. Yeats' work on Hydrocephalus—a boy nine years old.

[3] Clinique Médicale, p. 247.

Diagnosis.—1st. Congenital hydrocephalus has to be diagnosed from encephalocele and perforating fungus of the dura mater. In encephalocele the feel of the swelling is doughy and elastic, not fluctuating; it is local and not general, and it is not transparent. In cases of fungus of dura mater, which has perforated the cranium at birth, the general size of the head is not affected, the perforated spot can be easily detected, and it is over the central parts, not near the sutures or fontanelles; the mass feels doughy, elastic, quasi-erectile, and when it is compressed, symptoms of irritation are produced. Acquired hydrocephalus has also to be distinguished from a merely excessive development of the head apart from any disease. The absence of all cerebral symptoms is sufficient in such cases to establish the diagnosis. Sometimes hydrocephalus may be suspected where none exists, because of the disproportion between a small, emaciated, triangular face and largely developed skull. Rilliet and Barthez candidly confess to an error of this kind. 2d. From abnormal thickening of the bones of the skull, which sometimes obtains in rickets. In such cases the diagnosis may be made by a careful inspection and palpation of the bones of the head. The development of the skull is not uniform; it seems as if flat bumps had been superadded to the centre of the frontal and parietal bones, and we can detect with the finger the exact spot where the bone begins to thicken.[2] The swelling of the articular ends of the bones of the limbs, which is characteristic of rickets, will at once awaken suspicion, for rickets and hydrocephalus do sometimes co-exist. 3d. From hypertrophy of the brain. This is an exceedingly rare affection, in which the head enlarges without exhibiting any symptoms at first; and when these show themselves after a time, they run an acute course which soon terminates in death.

Treatment should be persisted in for a long time, without the adoption of any violent measures. The plan recommended by Professor Gölis, of Vienna, seems to be one of the best. He advises the head to be shaved, and a scruple or two of mercurial ointment, mixed with ointment of juniper berries, to be rubbed on the scalp twice a day. The child should wear a woollen cap, to prevent the risk of the perspiration being checked by the cold air. From a quarter to half a grain of calomel should be administered twice a day; if it purges too much, the inunction of mercurial ointment must be alone employed. This treatment is to be persevered in for thirty or forty days, when, if there be some improvement, the remedies may be gradually diminished; but the cap is to be worn after the inunction has been discontinued. If there be no marked improvement after six or eight weeks, some diuretic, acetate of potash or squills, for example, may be added; and a couple of issues may be inserted in the occiput. Blisters to the nape of the neck may be advantageously substituted for these. Whenever there is heat of head, and the child grows fretful, restless, and irritable, a couple of leeches behind the ears will be found of service. Gölis affirms that under this plan of treatment he has known the circumference of the head decrease by half an inch, or an inch, in a period of six weeks to three months. He thinks that convalescence, when once begun, may be accelerated by small doses of quinine. Dr. Gower's plan of treatment, which is said to have been successful in many cases, consisted in giving ten grains of crude mercury mixed by rubbing with about a scruple of manna and five grains of fresh squills. This was one dose, and it was to be repeated every eight hours. The medicine induced great prostration of strength, loss of flesh, and profuse action of the kidneys, without ptyalism.

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[1] In Rokitansky's Pathologische Anatomie, 1st edit. vol. ii. pp. 749—769.
[2] Rilliet and Barthez.

Chronic Hydrocephalus has been treated by two *mechanical* means: by bandages and tapping. Bandaging, which has been particularly advocated by Mr. Barnard, of Bath,[1] seems to be chiefly useful in pale flabby children, whose bones are loose and yielding: strips of plaster, about three-quarters of an inch wide, are made to encase the head; they are to be applied circularly, transversely, and diagonally Trousseau, who was at one time an advocate of this plan, has given directions for properly carrying it out in the *Journal de Médecine* for April 1843. But this eminent practitioner had good reasons for changing his views, and did not latterly advocate this plan. In his *Clinique Médicale,* second edit. p. 250, he says that he has given it up completely, since a child aged five months, whom he treated in that way, died suddenly on the fluid making its way through the æthmoid bone and the nasal fossæ.

The second mechanical mode of treatment, namely, by tapping the skull, and letting out the fluid accumulated in its interior, has been opposed by such men as Gölis, Richter, and Dupuytren. Dr. Conquest has been the greatest advocate of the operation in this country, and a paper on the subject may be found in the *Medical Gazette* for March 1838. Sir Thos. Watson gives the sanction of high authority to the procedure; and although Dr. West speaks rather doubtfully on the subject, yet he does not regard the operation as unjustifiable in some cases; when, for instance, there is good ground for believing that the hydrocephalus is external, or where the enlargement of the head has not been attended by indications of active cerebral disease. The operation itself does not seem to be attended with any very great immediate risk of life, if performed carefully. The best spot for puncturing the skull is about an inch, or an inch and a half, from the anterior fontanelle, near the edge of the coronal suture, taking care to avoid the longitudinal sinus, and some of the large veins which empty themselves into it. The trocar should be a small one, and it should be introduced perpendicularly. The fluid should be let out very slowly, a few ounces at a time, and the skull supported by bandages, both at the time and subsequently. If the child turns pale and faints, a few drops of ammonia, or of brandy, will be found useful. If any inflammatory action should be set up a day or two after the tapping, cold lotions to the head and leeches behind the ears, and small doses of mercury, will be required. The administration of iodide of potassium internally, and of iodine lotions to the scalp, has been advocated by Trousseau; and, when more active measures may not appear justifiable, some hope in the way of arrest of the further progress of the disease may be entertained from the use of these remedies. In addition to them I have found great assistance from the use of syrup of iodide of iron, cod-liver oil given in small doses, and bone-earth. The dose of cod-liver oil should be limited to a teaspoonful, the object being not to increase, but to improve nutrition. The iodide of iron is usually very well borne by hydrocephalic children, unless there be a tendency to congestion, or to inflammatory action. Amongst the children of the poor the combination of the oil and the syrup of iodide almost always gives the most satisfactory results. Bone-earth mixed with fine sugar, administered with every meal, sprinkled on the surface of milk, or of other food, has appeared to me to possess a tonic action beyond that possessed by any chemical compound of the phosphates. It has an increased value in cases associated with rickets or imperfect nutrition of the bones; and a diet, of which lentil flour forms part, has appeared to me highly advantageous. Good food, given in limited quantities, and at small intervals, is absolutely necessary, and I object to the use of stimulants. When the patient appears faint and languid, beef-tea will prove a better and more permanent stimulant than wine or ammonia. The usual hygienic measures should be adopted,—warm clothing for the extremities, the head being kept cool. Bathing with sea-water is useful, taking care that the limbs are rubbed to produce warmth and redness of the surface after the bath. The patient should, if possible, spend the summer months on the sea-coast, or in some elevated district, and he should almost live in the open air.

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[1] Cases of Chronic Hydrocephalus, &c., by T. H. Barnard. London, 1839.

MENINGEAL HÆMORRHAGE.

By J. Spence Ramskill, M.D.

The term Meningeal Hæmorrhage is used to denote extravasation of blood either into the cavity of the arachnoid, or beneath this serous membrane, and into the meshes of the pia mater. Hæmorrhage occurring between the dura mater and the bones of the cranium is extra-meningeal; and as it is usually the result of a blow or a fall on the head, in which case it often takes place on the side opposite to that of the injury, by *contre-coup,* it comes within the province of the surgeon and not of the physician.

In his valuable work on diseases of the brain, Abercrombie, at p. 238, relates a most curious instance of "extravasation in a cyst, formed by separation of the laminæ of the dura mater, from rupture of the middle meningeal artery." The patient, a man aged forty-eight, about the 12th of November, 1814, was assisting a neighbour to carry a heavy load up a high stair, when he felt a sudden attack of headache. He was from that time troubled with headache and giddiness, increased by stooping; and after these symptoms had continued rather more than a fortnight, he became sensible of some imperfection of vision. When seen by Dr. Gairdner, on the 2d of December, he complained of violent headache. The pulse was forty in the minute, and feeble. The pupils were at this time sensible to the light, but after a few days became insensible. He sank very gradually into coma, without any remarkable symptom, and died on the 13th. *Inspection:*—On the left side of the head, a cyst was found in the course of the middle meningeal artery, occupying the region of the lower part of the parietal and upper part of the temporal bone. It was formed by a separation of the laminæ of the dura mater, and contained about four ounces of coagulated blood. The portion of the dura mater forming the cyst was considerably thickened and very vascular. There was a depression on the surface of the brain, corresponding to the cyst, and the ventricles contained a considerable quantity of serous fluid. There was no other morbid appearance.

True Meningeal Hæmorrhage is an affection which is found generally at the two extremes of life, in infancy and old age. It occurs in newborn infants, after severe and protracted labours, and, from the discoloration of the skin attending it, is often mistaken for cyanosis. It may be distinguished from this malformation, however, by the absence of cardiac murmur, which is almost always present in the latter.

The blood may be diffused, as we have said, into the arachnoid sac itself, or under it, and in the pia mater. A third variety has also been described, in which the blood is said to be effused between the dura mater and the arachnoid; but recent researches have made it more than doubtful that the extremely delicate visceral layer of the arachnoid can be separated without being torn from the dura mater; and Baillarger[1] has shown that the error arose from the rapid formation of a false membrane resembling the arachnoid, which isolated the effused blood. An instance of this variety, of traumatic origin, is related by Sir Robert Carswell. A man fell on his head, was stunned for some little time, but afterwards went to work as usual. Three weeks afterwards, he applied to a hospital, but was refused admission because he had no fever, and he was suspected of malingering. On leaving the hospital he drank some hot spiced wine on his way home, became delirious, and died in thirty-six hours. A post-mortem examination showed six ounces of blood effused between the dura mater and the arachnoid, part of which was in a coagulated, and part in a fluid state.

Two cases of the same form of hæmorrhage, but of spontaneous origin, are given by Andral in his "Clinique Médicale," occurring in two men, aged respectively seventy and seventy-three. Other instances are recorded by Rostan,[2] Blandin, Ménière,[3] Cruveilhier.[4]

Hæmorrhage into the arachnoid cavity.—This may be traumatic or idiopathic. When the latter, the blood accumulates in the cavity of the arachnoid, and is equally diffused over the brain, not accumulated at the base. It is generally more fluid anteriorly, and more coagulated posteriorly. The arachnoid and dura mater are coloured by imbibition. After a time, the blood is enveloped in a pseudo-membrane, and in old cases cysts are found with yellowish contents and smooth walls; in some cases the two layers of the false membrane are found agglutinated, leaving no doubt as to the possibility of a perfect cure occasionally being made. With regard to the source from which the blood comes, there exists a discrepancy of opinion. According to most authors, the extravasation results from the rupture of a blood-vessel, but Prus[5] maintains that intra-arachnoid hæmorrhage is always the result of exhalation. Hæmorrhage by exhalation is, however, a pathological phenomenon not accepted nowadays, and for which cases of molecular rupture of blood-vessels used to be mistaken.

SYMPTOMS.—In persons of advanced age, there are sometimes certain premonitory symptoms observed in the shape of drowsiness, vertigo, general malaise, diminution of motor power, loss of speech, &c. All these symptoms do not show themselves in the same case, but one or other of them is generally present. Cephalalgia is a symptom which is usually met with in old people when hæmorrhage has occurred.

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[1] Baillarger: Du Siége de quelques Hémorrhagies des Méninges: Thèse. Paris, 1837.

[2] Rostan: Recherches sur le Ramollissement du Cerveau, p. 396.

[3] Anatomie Topographique. Paris, 1834.

[4] Anatomie Pathologique du Corps humain, livres vi. viii. xvii.

[5] Prus: Mémoire sur l'Apoplexie méningée (Mém. de l'Acad. Royale de Médecine), Paris, 1845, t. xi. p. 18.

Some authors regard the false membranes as being of inflammatory origin, and as the first step in the morbid process—the hæmorrhage being only the second.[1] There may be such cases, doubtless, in the adult and the old, but that they are very rare, in children especially, is sufficiently proved by the suddenness of the symptoms.

There may be sudden paralysis of motion on the side opposite to that of the extravasation; and when this is considerable in amount, both sides of the body may be affected, or paralysis may begin in one side and extend to the other. Sensation is rarely affected. Motor paralysis is not a constant symptom, and deviation of the tongue and of one angle of the mouth, and strabismus, are of very rare occurrence in adults, whilst they have never been observed in children: according to Legendre,[2] paralysis occurs only in one out of nine cases; Rilliet and Barthez say, in one out of seventeen.[3]

Contractions, rigidity of the limbs, and convulsions are, on the other hand, almost always present. There is at first somnolence, which gradually merges into coma; and this, when once established, persists, as a rule, unto the end. About the third or fourth day of the attack, there is intense fever lighted up, accompanied by the other symptoms of meningitis.

The course of the disease is exceedingly irregular; death may take place early or not until the end of a month. The prognosis is not necessarily fatal, and according to Legendre serous cysts may be formed, which give rise to a form of chronic hydrocephalus. Hæmorrhage may also occur beneath the arachnoid, between it and the pia mater. In some cases, it may take place suddenly; in others, it may be preceded by some headache, drowsiness, redness and heat of the scalp and forehead. When the extravasation has taken place there is generally headache, but not very acute, and having no fixed seat. Paralysis of motion is rare, probably from the thinness of the layer of blood effused. It has been said that when the blood is derived from a ruptured artery, motor paralysis is more apt to occur than when it proceeds from a ruptured vein, the difference being explained by the rapidity with which the blood escapes from the artery, and the incidental shock to the brain. Sensibility is not affected as a rule. The intellectual faculties are merely enfeebled, not perverted. After a time coma sets in, which persists until death.

On inspection after death, which seems to be an invariable termination of the disease, the blood is found in a liquid state, showing no tendency to coagulate, or to form pseudo-membranes. From the fact that no old cysts are ever discovered between the arachnoid and pia mater, it is inferred that sub-arachnoid hæmorrhage is invariably fatal. In his memoir on "Diseases of the Lateral Sinuses," Tonnelé[1] has related instances of rupture of a sinus giving rise to sub-arachnoid extravasation of blood. In a case reported by Dr. Mullar,[2] the blood came from the right lateral sinus, which was ruptured at its point of entrance into the torcular Herophili: death occurred in twenty-four hours, the symptoms preceding it having been those of cerebral hæmorrhage. According to Aitken, arachnoid hæmorrhage occurs when the extravasation bursts through the pia mater and arachnoid into the space between the membranes; and he says such an affection cannot be distinguished from ventricular extravasation. If, however, the extravasation is immediately arachnoid at first, and of limited extent, it may be approximately diagnosed: first, by the nature of the symptoms having partaken of meningeal inflammation, such as by severe pain in the head, with impaired intelligence and loss of power of movement; second, the attack is less sudden than in cases of congestion or of cerebral hæmorrhage, and the symptoms are progressively developed.

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[1] Consult Virchow: Die Krankhaften Geschwülste, Berlin, 1863, p. 140; and Lancereaux in Archives générales de Médecine, Paris, 1862, pp. 526—679, and 1863, vol. i. p. 38.

[2] Legendre: Mémoire sur les Hémorrhagies dans la Cavité de l'Arachnoïde (Recherches anat. path. et clin. sur quelques Maladies de l'Enfance). Paris, 1846, p. 130.

[3] Maladies des Enfants, p. 257.

The following are the combinations of symptoms which indicate sub-arachnoid hæmorrhage:—

First.—Complete and profound coma without paralysis, or with general paralysis slightly developed.

Second.—Complete loss of consciousness without paralysis, but combined with rigidity or clonic contraction of limbs.

Third.—Paralysis of hemiplegic distribution, as regards the limbs; but without deviation of the features, the muscles of the face not being implicated.

Fourth.—An apoplectic attack without anæsthesia.

Fifth.—Imperfectly developed coma with general paralysis.

Sixth.—An apoplectic attack, of which the symptoms are somewhat interchangeable or remittent.[3]

The TREATMENT of arachnoid hæmorrhage must be guided by symptoms present. When there is perfect coma with full, hard pulse, which is possible, a hot head, flushed face, turgid veins of the neck, and a hot general state of surface, with a slow, deep respiration, we may open a vein and take away ten ounces of blood with advantage; but in by far the majority of cases there will be no such opportunity. If the tendency be to death by syncope, the pulse small or feeble, the surface cold, the face pale and head cool, if there be signs of disease of the aortic or mitral valves, of kidney disease, or a general appearance of anæmia, we should do mischief by abstracting blood. The administration of a turpentine and castor-oil enema, and the application of an ice-cap or cold lotion to the shaved scalp, will generally limit our power of treatment, until the period of shock has passed away, or until consciousness has returned. Then the propriety of applying leeches must be measured by the degree of pain in the head, and of heat of the scalp. In the majority of cases even this will be found unnecessary. Free purgation will always be advisable, and the continued application of cold to the head. After a time, if the patient survives, the continued administration of the iodide of potassium promises the most hope of good. With respect to the abstraction of blood, it is right to say that the most eminent authority in Paris, Professor Trousseau, never saw any reason to order it. He denied the slightest advantage to be gained by it, either in arachnoid or in any other cerebral hæmorrhage.

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[1] Journal hebdomadaire de Médecine, Paris, 1829, tome v.
[2] The Lancet, June 1849.
[3] Reynolds: Diagn. of Dis. of Brain, &c., p. 101.

ADVENTITIOUS PRODUCTS IN THE MENINGES.

Under the heading of Syphilitic Meningitis the presence in the dura mater of so-called gummata has been adverted to. Calcareous deposits are also found sometimes in the substance of the dura mater; in some cases the falx cerebri has been found completely ossified.

HÆMATOMA OF THE DURA MATER is hardly recognisable during life. The symptoms, according to Aitken, extend over several months, and consist in general weakening of memory and of intelligence, the occurrence of giddiness, and local pain in the head. A chronic form of idiopathic inflammation of the dura mater is set up. At a later period an aggravation of all the symptoms occurs, with transitory losses of consciousness. Somnolence and apathy prevail, and generally one-sided paralysis of the extremities, which may soon disappear; eventually the case terminates with symptoms of apoplexy. On post-mortem examination, sanguineous and flattened masses, composed of fine layers of fibrine, spread to a greater or less extent over the dura mater, are discovered, accompanied by small extravasations which are converted into pigment. By repetition of the process, numerous layers come to be deposited one on the other. Numerous and larger blood-vessels form in these layers; and from these vessels renewal of the hæmorrhage occurs (Virchow, Weber). The lesion is sometimes described as due to intra-meningeal apoplexy, with false membranes on the dura mater; but the false membranes, which are the result of chronic inflammation, precede the apoplectic phenomena. The hæmatoma often attains considerable size. It may be four to five inches long, by two and a half broad, and one-half to three-quarters of an inch thick. It is generally of a flattened circular form, with a central elevation. The long diameter is parallel to the falciform process. The tumour generally occurs on one side only, or if bilateral, one is more developed than the other. The affection occurs only in the adult, and usually after the age of fifty. It has been clearly made out that the hæmorrhage which gives rise to the formation of the hæmatoma takes place between the layers of false membrane, and becomes encysted there. Treatment must be tentative. Iodide of potassium is our chief remedy. Any symptoms which indicate a recurrence of the chronic inflammation must be met by the application of cold to the forehead, by purgatives, and revulsives. If we can succeed in preventing the recurring attacks, there is fair ground for believing the newly-formed membranes may undergo a retrograde change and finally disappear.

Tumours have not been unfrequently found springing from the dura mater, varying as to their character and the nature of their contents. They are sometimes *fatty* and *encysted*, and have been known to contain hair; and Morgani (in Epist. Anat. xx.) speaks of an adipose tumour with hair in the substance of the tentorium. But sometimes also the tumour is of cancerous nature, constituting what has been termed "fungus of the dura mater." The celebrated French surgeon, Louis, has written a most important series of essays on the subject, published in "Mém. de l'Académie de Chirurgie," vol. v. p. 1, Paris, 1774. The cancer may be of the *encephaloid* or of the *scirrhous* variety: the former is the more frequent of the two, and indeed, when the tumour has made its way outwards by perforating the bones of the skull, it has often been mistaken for hernia cerebri. The tumour may spring from the outer or the inner lamina of the dura mater, and in some cases there may be tumours co-originating on both surfaces of the dura mater, as in cases reported by Chelius and by Dr. Bright. When they spring from the inner surface of the dura-mater, they have a tendency to grow inwards and depress the surface of the brain. But in comparatively rare cases they press on the skull, cause absorption of its substance, and protrude externally. In one case, described by Cruveilhier, protrusion had actually occurred; in another, perforation was in progress. In connexion with the dura mater are sometimes also found *fibrous* tumours, which, on microscopical examination, are seen to possess the same curvilinear stromal arrangement as the common uterine fibrous tumour.

The bony plates found on the inner side of the dura mater have by some been regarded as growth belonging to the arachnoid, but that they are not so is sufficiently proved both by the position in which they are found, and by the property of periosteal tissues, to which class the dura mater undoubtedly belongs, to generate bone.

In connexion with the choroid plexus (that intra-ventricular appendix of the pia mater) the adventitious products found have been indurated yellow bodies, the remains of former hæmorrhagic effusions, and more frequently round or oval bodies of a yellowish tinge, apparently formed of concentric laminæ, which only become more apparent on the addition of acetic acid. They are generally microscopic, but sometimes accumulate into masses of the size of a pea or small nut. They have been called by Virchow *corpora amylacea*, and by Dr. H. Jones *concentric corpuscles*.

Small *cysts* are also pretty frequently found on the choroid plexus, which have by some been erroneously spoken of as hydatids, but there is no evidence to show that they belong to those parasitic formations. They rather seem to be due to a condensation of the epithelial covering of the plexus, and an accumulation of fluid beneath it, limited by an effusion of plastic matter.

CONGENITAL MALFORMATIONS OF THE MENINGES.

The dura mater is the one generally affected, and such cases are of extremely rare occurrence. Sometimes the falciform process is entirely or partially deficient, as is also the tentorium.

The falx is, of course, absent when the cerebrum is undivided ; or if the cerebrum be single in front and divided behind, the falciform process begins to appear where the division is, namely, at the coronal suture. In monsters, in which the posterior lobes of the cerebrum are deficient, the tentorium is also deficient. In a girl, seventeen years old, who was idiotic and motionless from birth, the hinder part of the tentorium was deficient.[1] The Reports of the Pathological Society, 1847 and 1848, p. 178, contain the account of a very rare defect of the falx cerebri exhibited by Mr. Shaw. Dr. Bright also gives a similar case, in which no trace of the process was visible anterior to the tentorium, and it was assumed that the defect, which occurred in a lady of thirty years of age, had existed from birth.

[1] Gilbert, in Edinburgh Medical and Surgical Journal, No. 95, April, 1828.

CONGESTION OF THE BRAIN.[1]

By J. Russell Reynolds, M.D., F.R.S. and
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Under this name there are to be included several forms of disease very different from each other in the general character of their symptoms. In one of these the patient is feverish, and his attack is sometimes regarded as "brain fever;" in another the case is described as an "apoplectic" or "paralytic stroke;" in a third, as a fit, or seizure of "convulsions;" and in a fourth, as an attack of "delirium," or of "wandering." Patients taken with symptoms of disturbance, in any one of these forms, often die; and upon post-mortem examination there may be found but one departure from healthy appearance of the brain, viz., congestion. We cannot but suspect that in many cases there have been alterations of nutrition which have escaped our notice, and that the locality and nature of such alterations have determined the form that the malady has taken. We may infer in others that, if the congestion has been the sole cause of symptoms, the character of the symptoms which accompanied it was determined by the situation of the excess of blood. But we cannot yet demonstrate the truth of these surmises or inferences, for we cannot see the brain while the symptoms last, and the most characteristic often pass away before the patients die. The premonitory, and even the earlier developed symptoms of cerebral congestion may be closely similar in many cases; they then speedily pass into one or another of the several groups enumerated above, and from the special characters which they then present the cases derive their names: but if the morbid state continue, and advance towards a fatal issue, the distinctive features of these several forms pass away; convulsion, delirium, and febrile action cease; and patients, who a few days before presented very wide symptomatic differences, look much like each other, and die in a similar manner. Such being the case, we should expect to find the traces of that which existed only a short time before death, and to fail in discovering evidences of those localized changes which must have determined the character of the previous symptoms.

It will be convenient therefore to describe first the premonitory symptoms of cerebral congestion, viz., those which are or may be common to its several forms; then to detail under four distinct categories the developed symptoms, with their modes of termination in recovery; and lastly, to describe again generally those which are final, and into which any one of the four varieties may pass by a rapid or a gradual progress.

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[1] The sections on Pathology and Morbid Anatomy are written by Dr. Bastian.

SYMPTOMS.—A. *Premonitory.*—There is often a mixture of two classes of symptoms,—those which indicate both over-action and the reverse; sometimes the two co-exist, at other times they alternate; in one case the former group is predominant, in another the latter; whereas in a few all the symptoms tell in the direction either of inaction or of undue excitement. It is possible sometimes to foretell, from the nature of these symptoms, the form which it is most likely that the disease will hereafter assume; but such forecast is uncertain in all cases, and useful in only a very small proportion.

The *mind* is changed in such manner that there is diminished intellectual power; thought becomes confused, and memory treacherous; the individual may be irritable, "put out about little things," worried, fanciful, peevish, or depressed; sleepy, and especially so after meals; at times indifferent and sluggish; he complains that he "cannot think," and that the forced effort to do so makes him worse; he talks at random, using wrong words—sometimes noticing his blunder, correcting it, and expressing his annoyance; sometimes not observing that he was wrong, and being greatly annoyed with any one who should attempt to set him right. He is usually worse after being in the recumbent posture, and after sleep; his sleep is heavy, and disturbed by dreams and nightmare; sometimes there are transient delusions—one person is mistaken for another—the past and the present are curiously intermixed, and the conversation is like that of a dream, a dream which goes on while the patient is awake, but from which he may be awakened still further by a loud voice or any other strong appeal to the senses.

The *senses* are dull: hearing is defective, and there are rumbling "noises in the head;" the sight is dim, and "black specks" appear before the eyes; sometimes diplopia is present; there is giddiness, and a feeling of oppression and fulness in the head, with "stupid headache," made worse by lying down. There is rarely "pain" in the head, but, as the patients say, "a confused, uncomfortable feeling." The limbs feel heavy, and there is often numbness or "pins and needles" in the toes and fingers. These sensations come and go, but between the periods of their recurrence there is a sense of general discomfort which it is often quite impossible for the patient to describe. Often it is that of "oppression about the breathing;" and great difficulty from this source is experienced in walking upstairs, uphill, or even a little more quickly than usual on level ground. Sometimes "feelings of faintness" are complained of, and with them nausea and increased vertigo.

The power of *movement* is diminished, and with it yet more notably the readiness of action. The limbs are dragged along sleepily, or sluggishly; the step loses its elasticity, is shorter than in health; the "general bearing" is changed; and sometimes, but rarely, the alterations in power and activity are observed on one side of the body more distinctly than on the other. The patient simply leans forward, and appears weak and lethargic; or he may lean to one side, hold one shoulder half an inch or an inch higher than the other when standing, and when sitting, collapse, as it were, on the lower side. Friends of such patients say, "He seems to go down on this side," and, "We are afraid that he will fall off his chair, or off the pavement," but the physician may observe no paralysis; for the stimulus given to voluntary effort by his presence is often enough to remove the trifling want of symmetry. The features are regular, the tongue is straight in its protrusion, and the grasp of the hand and the movements of the feet are as pronounced on the one side as on the other.

There are *other* symptoms than those of direct change in the nervous functions, such as redness, and often dusky redness, of the lips, conjunctivæ, face, and scalp. The head is hotter than the cheeks, the jugular veins are distended, and the neck appears thick. On stooping, sneezing, or coughing, the veins of the forehead are too full, and the beat of the carotids is too distinct. The pulse is slow and laboured, or quick and feeble; the tongue is foul, the urine small in quantity, and often loaded with lithates; the bowels are confined, and the extremities are cold. The heart may be found dilated, and there may be tricuspid regurgitation, shown by the pulsation of the jugulars, and systolic murmur at the ensiform cartilage.

These premonitory symptoms may exist for very variable periods of time; may appear and disappear; or may gradually increase and pass into one or another form of malady already hinted at, but now to be described.

B. *Developed Symptoms.*—1. *Apoplectic form.*—The attack usually takes place during some muscular exertion, such as lifting a heavy weight, blowing the nose, coughing, sneezing, straining at stool, or stooping to pick up something from the floor. Sometimes it cannot be traced to any one of these. But it rarely occurs during sleep; patients do not wake up and find themselves in a state of what is called "congestive apoplexy." They are more commonly doing their ordinary work, or trying to do a little more than they are able to accomplish, when the attack is made. Consciousness, sensation, and power of motion seem to be lost, and the patient is said to have "an apoplectic stroke;" but these faculties are not altogether lost, or if they are, it is for a few moments only, and the physician usually finds the following conditions:—

The *mind* is not in complete abeyance. There are indications that the patient knows, although but imperfectly, what is said to him; he makes some attempt to respond to questions, and to do what he is asked to do. He starts at a loud and sudden noise, looks round him, and gives signs of annoyance when he is disturbed. If at the moment of seizure he should appear to be in profound coma, this coma is of short, almost of momentary duration, and soon there are signs of returning consciousness: there is confusion of thought, bewilderment, and dulness of apprehension, passing sometimes into a mild delirium, but more often into a heavy sleep.

The *senses*, obtuse for a moment, are rapidly restored to a certain point. The patient shrinks from strong light, groans when pinched, starts when spoken to, but yet takes little or no notice of ordinary impressions.

The power of *motion* is so diminished that the patient falls down, and the limbs when raised fall heavily. All of them appear equally weak; but, in a few moments, occasional voluntary movements may be seen in them, and these are commonly more distinct on the one side than on the other. There are slight twitchings of the muscles, but there is no rigidity. The features are usually symmetrical, or if drawn to one side are speedily set straight again. There is no stertor in the breathing; the speech is clumsy, the words are clipped, and wrong words are used; but this is for a short time only, and the sphincters very rarely fail in their action.

The *pulse* at the moment of attack is sometimes suspended at the wrist, and the *breathing* is arrested; but soon the pulse is felt to be heavy and laboured, and the respiration becomes tumultuous; and again, in a few seconds, both pulse and respiration go on as they did before. There is an exaggeration of the previous vascular fulness of the face, neck, and head; sometimes a bloodshot eye, or epistaxis; not unfrequently vomiting, with apparent faintness and a condition of collapse.

The symptoms of a first attack usually abate quickly; they may last for a few minutes, or for several hours, but most commonly they disappear within an hour; and the patient, although languid and perhaps alarmed, may feel better than he has done for some days before. Upon the repetition of seizure, however, the duration of symptoms is prolonged; the recovery of consciousness, sensation, and movement is less complete; drowsiness is more marked; and if there be some momentary awakening, it is momentary only; fresh attacks supervene, and each leaves the patient lower than he was before.

The apoplectic form of cerebral congestion is most common in advanced life, and has usually been preceded, and that to a marked degree, by the "premonitory" symptoms that have been described.

2. *The convulsive form.*—The paroxysms that occur have the general features of epilepsy (see Volume I.); but they differ from the attacks of that disease in their general history and mode of onset. Congestive convulsions may occur at any period of life, but they are most frequently met with at the time of full maturity, or when that stage is passed. There are usually the premonitory signs of congestion, but these may be very slight; there may be no forewarnings, and the patient may be seized during sleep, or while making some unaccustomed effort. When the attack occurs

during sleep it is difficult, and sometimes impossible, to say in what manner it commenced; but when it has come on while the patient is awake and friends are about him, it has been usually observed that much discomfort has preceded it, for a few seconds, minutes, or hours. A tight cravat, worn while making some undue exertion; a sudden alarm; or an indigestible meal, rapidly swallowed, may be the immediate antecedents. The patient, more or less suddenly, becomes confused, then apparently half unconscious, makes some unintelligible sounds, turns red and then blue in the face, staggers for support, looks round him wildly or imploringly, and then sits down, or falls down, convulsed, and a paroxysm, epileptiform in character, supervenes. From this he recovers partially, exhibiting great confusion of mind, headache, muscular feebleness, and sometimes partial paralysis of one side, or of one limb. The attack is occasionally followed by quasi-maniacal excitement, lasting from half an hour to three or four hours; after which, the patient becomes exhausted and falls into a heavy sleep. From this state he may recover, or during sleep a second or third attack of convulsions may come on.

When congestion of the brain has assumed this convulsive form, the patients, so far as my own experience extends, have usually been in middle life, and have recovered. But in other instances, when the age has been further advanced, the attacks have recurred more frequently, the intervals between them have become of shorter duration, and the patients—less sensible and less reasonable after every paroxysm—have presented the appearance of those whose attacks have been apoplectiform at their commencement.

3. *Delirium* may be the most marked symptom of congestion of the brain in certain cases. This is observed almost exclusively in those who are of an advanced age, but it is not absolutely limited to the period of senility. It may occasionally be met with in middle or even early life, and is then commonly accompanied by some change in blood-quality. The attack may come on suddenly, may be induced by a fall or a fright, but when occurring spontaneously is first observed towards evening. Sometimes the attack is preceded by "depression of spirits;" the patient, after some hours or even days of undue taciturnity, becomes cheerful, or gay, and hilarious; he talks loudly and incoherently, but rarely exhibits any violence. He gets out of bed, wanders about his room or ward, opens drawers, puts on his dress, and is bent upon doing something which he cannot explain, or which, if expressed in words, is unnecessary, unaccustomed, and absurd. He is under a delusion, of no fixed character; and can usually be directed and managed without much difficulty. Sometimes, and this is especially observed in the aged, there may be hysterical crying; or, still more rarely, great irritability of temper and some attempts at violence. The latter occur almost exclusively as the result of bad management and rough thwarting of the delusive purpose.

The patient may complain of pain in the head, or of uneasy sensations in the limbs; and there may be twitching of the muscles, or weakness of the extremities. But none of these are complained of while the delirium lasts, although weakness and clonic spasm may be observed at the time of its occurrence. It is when the delirium has completely or partially subsided that these things are noticed, and that the general phenomena of cerebral congestion, viz. those which are described as premonitory symptoms, may be observed. Durand Fardel states that it is common to find "a mucous secretion, clear and viscid, produced on the eyelids, or in the interior of the mouth, and sometimes in extraordinary abundance, running over the whole face;"[1] but this is very common in other diseases of old people, and has no special relation to mere congestion of the brain.

The recurrences of delirium may be very frequent or very occasional: some old people present them nightly for many weeks, and know nothing about them on the following days; while others exhibit them after much longer intervals, and only when "upset" by the little occurrences of the day. The tendency, however, is towards increase—not so much in degree as in persistence of mental change—and the patients become gentler, but less rational. The mind is weakened at each onset of delirium, and does not recover itself; there is drowsiness in the daytime and wandering talkativeness at night; but the intellectual powers are seen to be failing day by day; the physical energy diminishes, and the patient keeps his bed, and gradually passes into the state hereafter to be described.

4. *Febrile form.*—In the earlier periods of life, and especially in infancy and childhood, congestion of the brain may occur with marked elevation of temperature, a dry skin, thirst, and the restlessness and malaise of a pyrectic state. There is headache, not of great intensity, but of dull, oppressive character; the head is unduly hot, the cheeks and conjunctivæ are flushed, while the extremities are cold; the mental faculties are obscured, and the sleep is broken by dreams or transient and mild delirium. Usually there has been some distinct cause for such disturbances; there is no marked prostration, no initial rigor; there may be some vomiting, but it is not persistent, nor are the bowels obstinately confined; there is no photophobia, no intolerance of sounds, no eruption on the skin; the secretions may be foul, but they present no indications of organic disease; and the patients usually recover speedily. Recovery is, however, not always observed; the distress may persist; there may be, alternately, convulsions and delirium, or there may be the changes from over-excitement to drowsiness, the latter gradually becoming relatively more marked, until the patient passes into a state of stupor from which he may never rally.

C. *Final Symptoms.*—Under whatever form congestion of the brain may primarily appear, its tendency, unless speedily recovered from, is to produce a condition of torpor and inactivity. The mind becomes a blank; there is sometimes profound coma, stertorous breathing, and involuntary evacuation of both bladder and rectum; sensibility both general and special is lost, and voluntary muscular power reduced to a minimum. Convulsions may occasionally disturb the calm, or there may be fitful and momentary muttering of unintelligible sounds, but usually, in this latter stage, the patient lies quietly, with laboured pulse and breathing, and with flickering contractions of the muscles of the limbs, until he dies.

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[1] Maladies des Vieillards, p. 27.

CAUSES.—Among the predisposing causes must be reckoned such physical conformations as should impede the return of blood from the head, and the most important of these is a morbid condition of the heart. Dilatation of the right side of the heart, with loss of both power and valvular competency, are commonly found, during life and after death, in those who succumb to cerebral congestion. It is in old age that such changes are usually discovered, and hence advanced age appears as a predisposing cause. It is more common to find severe cerebral congestion in men than in women. Sedentary occupation and shortness of neck have been reckoned among the predisponents, but I think with insufficient reason; for attacks of cerebral congestion often occur in those of active habits and of healthy build, and indeed sometimes the worst forms of seizure that I have witnessed have been in persons of great mental and physical activity, in those who have been overwrought, and who have continued in forced exertion beyond the bounds of reason and habitual practice.

The determining causes are to be found in all those conditions which entail sudden changes in the circulation. These are exposure to extreme heat or cold, and especially to the direct influence of the sun's rays; blows upon the head or trunk; violent exertions, such as make it necessary to "hold the breath;" rarefaction of the air, such as is encountered in balloon ascents, and in some mountaineering expeditions; violent emotion, or prolonged mental effort; an overloaded state of the stomach, and this especially after undue abstinence; the ingestion of large quantities of alcoholic stimulants; a sudden change of posture, such as stooping or lying down with the head too low; and tightness of the dress around the neck.

Besides the so-called predisposing and exciting causes of cerebral congestion, there are two general conditions of the organism which may have some causative relation to the symptoms, although neither of the preceding words fully conveys the nature of that relation. A full-blooded, lax-fibred, and fat man, in middle age, represents one of these conditions: a thin, pale, wiry old person, with rigid vessels, is an example of the other. Both are prone to suffer from disturbances, irregularities, inequalities in the circulation; and in either there may be cerebral congestion in a grave or fatal form. If these conditions be regarded as "predisponents," it must be remembered that they have no special relation to this locality of congestion, and further that the mode in which the one operates is quite distinct from that in which the other leads to its results. In the former cerebral congestion is but part of a general condition, and some accidental posture may determine that the brain shall be the organ upon which the weight of the burden falls; in the latter, feebleness of circulation power, and locally increased resistance in the walls of vessels, may be the main factors in the production of such partial congestion of the brain as shall give rise to an apoplectiform seizure.

DIAGNOSIS.—Remembering the general character of the symptoms which were described as "premonitory," there can be but little difficulty in carrying the diagnosis up to a certain point, and in explaining them by the fact of congestion; but when the malady passes into either one of the four forms of "developed" symptoms, the diagnosis is sometimes difficult, and it is therefore necessary to consider it in detail with regard to each.

The *apoplectic form* of congestion resembles cerebral hæmorrhage, acute softening of the brain, urinæmia, and syncope. From hæmorrhage, it may be distinguished by the facts of its less sudden onset; its occurrence while the patients are awake rather than when asleep,—patients do not wake up in the morning and find themselves paralysed on one side, as they often do in cases of hæmorrhage;—the attack of "congestive apoplexy" occurs during the day, and its onset is marked by the absence of the phenomena of shock; by the equality of disturbance usually noticed in regard of mind, sensation, and motility at the commencement of the seizure,—each of them is affected generally, and to nearly the same degree, but in no one direction is there entire and absolute loss of function except for a few moments; by the subsequent relative proportion of symptoms, such for example as partial paralysis of all the limbs with imperfectly developed coma, a combination not to be observed in hæmorrhagic apoplexy; by the speedy restoration of the mental faculties; and by the equable and usually simultaneous removal of other symptoms.

From softening of the brain in its acute form, congestive apoplexy cannot be always distinguished at the outset, for in some cases of the former the attack is in reality due to the occurrence of the latter. The diagnosis can only be made after some little time has elapsed, and then it will turn upon a recognition of the following points:—In congestion the mind speedily recovers, in softening it does not; in the former there is widely distributed but imperfect paralysis, in the latter limited, but more complete, loss of power; in the one the patient is generally powerless, in the other he is hemiplegic; in the one there is flaccidity of muscle, in the other there is rigidity; in the former the premonitory symptoms have been those of congestion of the brain, in the other those of chronic disease elsewhere and loss of power.

From urinæmia, the attack may be distinguished by regard to the premonitory symptoms; by the absence of œdema of eyelids or of lower extremities; by the absence of albumen from the urine; by the absence of marked rigidity of muscles; by the nature of the coma, its momentary profundity, rapid diminution, and want of that peculiar character which often attaches to blood-poisoning, viz. its apparent profundity in strong contrast with the ease with which the patient may be awakened up to a certain point; and further by the absence of a peculiar variety of stertor, occasioned apparently in the mouth or at the palate.

From syncope, congestive apoplexy may be distinguished by an examination of the heart, and the pulse at the wrist, the carotids, and the temples; by the colour of the face and head; the premonitory symptoms, and the conditions which led to the attack.

The *convulsive form* of congestion may be confounded with epilepsy or with eccentric convulsions. From epilepsy the diagnosis may be made by a consideration of the previous history: in the one there have been the premonitory symptoms of congestion, in the other no such phenomena have presented themselves; in the former the patient is usually of middle or advanced age, in the latter he is young, and is either under twenty years, or has not far exceeded that period of life; in the one the period of most marked congestion is at the moment of onset of the seizure, in the other congestion of the face and head is most marked as the attack is passing off; in the former there may be some moaning sound, in the latter the "epileptic cry;" in the one there is the sudden onset of an acute disease, in the other the attack of a chronic malady.

From eccentric convulsions it is possible to distinguish congestive convulsions by regard to age and attendant symptoms. Eccentric convulsions are observed in infancy and early life, and when some definite source of irritation can be discovered in certain organs of the body; they are found most commonly in the weak, irritable, and nervous subject, and they are attended by no premonitory symptoms of congestion, and by little or no evidence of its presence during the attack. There is but trifling somnolency, and the seizures differ from those of epilepsy and of congestive convulsion in not passing through the stages which were described as proper to the former, and which are closely simulated by the latter.

Congestion of the Brain in the *form of delirium* is met with almost exclusively in old age; and it is necessary only to mention delirium tremens in order to prevent the possibility of their being confounded. From senile softening of the brain, when this is accompanied by recurrent delirium, the diagnosis may be made by regard to the intermediate state; for when only congestion is present the patient returns to his normal condition in the intervals of wandering, whereas when the brain tissue is undergoing degeneration, and is the cause of delirium, no such recovery is possible. There is, moreover, a progressive enfeeblement of all the nervous functions, and a general condition of depraved nutrition such as is not necessarily found in cases of congestion.

The *febrile form* of congestion may be distinguished from meningitis by the absence of acute pain, and of intolerance of sensorial impressions; by the milder character of the delirium, the dilatation rather than contraction of the pupils, the absence of persistent vomiting and of obstinate constipation, the generally milder character of the symptoms, and their early cessation.

Pathology.—The circulation through the cerebral vessels has been supposed to present certain peculiarities owing to the enclosure of the brain within an unyielding case, and its being, therefore, beyond the influence of atmospheric pressure. This was first alluded to by the second Monro. It was thought that no great alterations could take place in the total quantity of blood within the cranium at different times, although there might be an altered ratio as regards the respective amounts of arterial and venous blood. It was even held by Dr. Kellie, that in animals which have died from hæmorrhage there is no lack of blood in the brain; that where, on the contrary, we should expect to find a condition of cerebral hyperæmia, we do not meet with it; and that the quantity of blood in the cerebral vessels is not affected by gravitation, and thus is uninfluenced by the position of the head with respect to the body. These views were also supported by Dr. Abercrombie and by Dr. John Reid;[1] though they have been ably opposed by Dr. Burrows[2] and by Donders,[3] many of whose experiments go to establish the direct reverse of the results arrived at by Kellie. It seems by no means satisfactorily demonstrated that the contents of the cranium are so entirely removed from the influence of atmospheric pressure. Dr. Burrows says: "The numerous fissures and foramina, for the transmission of vessels or nerves through the bones of the cranium, appear to me to do away with the idea of the cranium being a perfect sphere like a glass globe, to which it has been compared by some writers." And the other dogma on which this hypothesis rests, and which Dr. Abercrombie supports when he says, "We may safely assert that the brain is not compressible by any such force as can be conveyed to it from the heart through the carotid and vertebral arteries," seems to be directly contradicted by a consideration of other facts.[4]

The observations of Robin,[5] and of His,[6] who have discovered a system of lymphatic sheaths inclosing spaces around the cerebral blood-vessels, are of great importance, and reveal a structural adaptation which seems especially calculated to permit of varying amounts of fulness of the cerebral vessels, within certain limits, without injury to or compression of the surrounding nerve pulp. Professor His has succeeded in injecting this system of perivascular canals, and has found them most obvious in the grey matter of both brain and spinal cord. He has found that the injections at first reach the surface of the encephalon and cord, and fill a vast system of lacunæ situated between the pia mater and the surface of the nervous centres; while, if pushed still further, he has found that they fill the lymphatics of the pia mater itself. Thus there is, as it were, a second series of vessels inclosing and surrounding with a fluid medium all the ramifications of the cerebral and spinal vascular system, whilst these two sets of vessels, containing and contained, are lodged in definite cylindrical canals permeating the nerve substance in all directions. The lymphatic sheaths are in contact with, though in general are easily separable from, the walls of these canals through the nerve substance. The diameter of the canal (and therefore of the lymphatic sheath) may be seen, in transverse sections, to be generally twice, and sometimes three or four times, as large as that of the contained blood-vessel. It will be easily understood that these two systems must have such a complemental relationship to one another, that an extra fulness of the one set of vessels will correspond with diminished fulness of the other set. That is to say, in order to make room for an increased amount of blood in the cerebral vascular system a corresponding amount of fluid must be driven out of the enveloping lymphatic vessels;[1] whilst, when the vascular supply is again diminished, a proportionate amount of fluid re-enters the cerebral lymphatic canals.

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[1] Physiolog. Anatom. and Path. Researches, No. xxv.

[2] Lumleian Lect. 1843, and On Disorders of the Cerebral Circulation, &c. 1846.

[3] Nederland. Lancet, 1850.

[4] Andral's Clinique Médicale.

[5] Brown-Séquard's "Journal de Physiologie," 1859, p. 527.

[6] Zeitsch. für wissen. Zoolog. 1865, Bd. xv. and The Journal of Anatomy and Physiology (Cambridge), No. 2, p. 347.

Thus, we believe that the amount of blood existing within the cranium may be subject to great variation, and that the peculiarities of the cerebral circulation have been much overrated.

The conditions capable of bringing about a state of cerebral congestion are very various, and so also is the degree of hyperæmia met with, and the extent of its diffusion over the encephalon. In one class of cases, the congestions seem to be most obviously mechanical phenomena, due to some impediment to the proper return of blood from the brain, owing either to diseases of the heart or lungs, to pressure upon the great veins by tumours, or to their obliteration by thrombosis. In other instances, however, the condition of hyperæmia seems a more purely vital phenomenon, as when it is the result of prolonged study and over-mental work, or when it has an irritative origin, and is set up around some old clot, bony exostosis, or adventitious product in the brain. Then, too, alcoholic intoxication, great elevations and alternations of temperature, exposure to the sun's rays in hot summer weather, and the suppression of accustomed fluxes, whether menstrual or other, are all looked upon as occasional causes of cerebral congestion. In connexion with inflammation of the meninges, congestion of the convolutional grey matter is doubtless the initial stage of what afterwards becomes diffuse superficial cerebritis. Well-marked Congestion of the Brain is also met with very frequently in persons who have died whilst suffering from symptoms of delirium or coma during the course of the acute specific diseases, and in whom there may be no trace of meningeal inflammation. This is more especially common in typhus fever. From observations which I have made on the bodies of persons who have died from this disease, and also from the minute examination of the brain of a man who died delirious whilst suffering from acute phlegmonous erysipelas of the head and neck, I have been led to believe that these minute and widespread congestions are often due to embolism or thrombosis of the minute arteries and capillaries of the brain.[1] Wide-spread obstructions in the small vessels, however brought about, would cause much of the propulsive energy of the heart to be wasted and a consequent lagging of blood in the venous radicles.

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[1] Cambridge Journal of Anat. and Physiol., No. 2, p. 351, note 2.

Cerebral congestion is very intimately related to cerebral hæmorrhage on the one hand, and to inflammation on the other. Hæmorrhage is most likely to be associated with the congestions of mechanical origin, especially if these are brought about rapidly; and although such cerebral conditions generally give rise to well-marked brain symptoms, still the groups of symptoms previously described are often related to congestions of a more active kind—such as are commonly spoken of as 'determinations' of blood to the head—and which may be said to commence rather on the arterial than on the venous side of the circulation. In these cases, perhaps by virtue of certain changes occurring in the nerve tissue itself, an increased flow of blood takes place to the brain, which may subside after a variable time and after the production of a certain set of symptoms, or which may occasion the death of the patient owing to the supervention of symptoms of a graver type. In certain other cases the congested condition of the membranes and cortical substance may gradually lapse over into a state of inflammation, and it will then be associated with tissue changes of a more marked character.

MORBID ANATOMY.—Congestion of the brain tissue itself is almost invariably associated with a similar condition of the pia mater, and the amount of cerebral congestion is often judged of, in a loose way, by the degree of fulness of the vessels of this membrane. What many persons would consider to be a state of congestion is, however, natural to the vessels in this situation. Hasty opinions on this subject should, therefore, be especially guarded against. This fulness of the vessels of the pia mater is most notable in the occipital region, whither the blood gravitates, for the most part, after death. Occasionally, however, as suggested by Laborde, this occipital congestion may take place during the last days of life, so as to place it in the same category with hypostatic congestion of the lungs.

In some cases, where there has been every reason to believe that a state of congestion existed during life, it must be confessed that little or no traces of it can be recognised after death; though, on the other hand, when it has existed for some time and has been carried to an extreme degree, or when it has been often repeated, undoubted evidences of the present or previous existence of such a condition may be met with. In a young and middle-aged subject, in whom no atrophy has taken place, but whose brain has been subjected to an extreme degree of congestion during life, the organ frequently seems, after removal of the calvarium, to be in a swollen condition. The dura mater is tightly stretched over the organ, and after its reflexion the convolutions appear broad and flattened, with sulci less obvious than natural, owing to the effects of pressure against the interior of the skull. Then, there is not only the usual fulness of the large veins of the pia mater, but also a more tortuous and even varicose condition of these trunks, together with a more complete injection and turgescence of the smaller vessels than is usually encountered. The membranes may be stripped off the surface of the convolutions without tearing the grey matter, and on section this appears darker than natural, and dotted with bloody points in the situations of its loaded vessels. The white substance also shows an abundance of a certain number of the red points, which are usually gorged vessels pulled out for a certain distance so as to lie on the cut surface. These are only comparative signs, however, and their true value must be estimated accordingly, since all intermediate conditions may be met with between the ordinary healthy amount of fulness and the most marked degree of hyperæmia. It is extremely difficult to draw the line and say what is morbid and what is consistent with health.

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[1] "On the Clogging of Minute Vessels in the Grey Matter of the Brain as a cause of Delirium and Stupor in severe Febrile Diseases; and on other Symptoms of the 'Typhoid State:'" Brit. Med. Journ. Jan. 23, 1869.

If, however, the congestions have been often repeated or have lasted for any length of time, microscopic examination does enable us to discover evidence of this. The capillaries, and more particularly those of the grey matter, become twisted and varicose, displaying partial dilatations, or real aneurysmal swellings, implicating either a part only of the calibre of the vessels, or dilating them in numerous adjoining parts in their whole extent, so as to constitute "l'état moniliforme" of Laborde.[1] But a still more certain mark of old congestion is afforded by the presence of a quantity of blood pigment (hæmatine) surrounding the vessels, though inclosed within the lymphatic sheath described by Robin. It is met with in the form of more or less rounded simple or molecular grains, mostly of large size. They may measure as much as $\frac{1}{2000}''$ in diameter. They are usually of a dark olive or amber yellow colour, and are sometimes composed of a number of minute pigment granules aggregated into small spherical masses. The pigment remains quite unaltered after the application of ether, alkalies, or the strongest acids. The crystalline form of blood pigment (hæmatoidine) is not met with, since this seems to be produced only in places where an actual extravasation of blood has taken place, whilst the pigment in the granular and amorphous condition seems to result from stasis of blood, and more or less transudation of colouring matter, or hæmatine, through the walls of the vessels into the surrounding lymphatic canals. It seems impossible otherwise to account for what I have seen. I have found, for instance, this matter in great abundance around almost all the small vessels and capillaries that were examined belonging to the brain and spinal cord of two individuals. Both were lunatics; the one an epileptic and chronic maniac, subject to paroxysms of great excitement, and the other a chronic maniac of the most violent and excitable disposition, whose fits of passion were both frequent and long-continued. It was during the examination of the brains of these individuals that this granular blood pigment, surrounding the vessels, first attracted my attention. I have since found that a similar condition had been noticed and described by Robin, and I can endorse his statement that a few such masses of pigment are usually to be met with, here and there, on the cerebral vessels of even young and healthy subjects. It is, therefore, the abundance of this matter only which is to be looked upon as an index of disease; and the duration of past congestions may be roughly guessed at by the more or less excessive accumulation of pigment around the vessels.

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[1] "Le Ramolliss. et la Congest. du Cerveau," Paris, 1866. These irregular aneurysmal dilatations of thin-walled capillaries must not be confounded with the distinct though microscopic aneurysms, occurring on some of the smallest arteries after they have undergone a process of fibroid thickening in different parts of the brain in old people. We have previously hinted at the occasional connexion between cerebral congestion and cerebral hæmorrhage, and now we may state that the links which bind the two together are frequently the aneurysms just mentioned. Congestion may have something to do with their formation, as it certainly has to do with their final rupture, leading to effusion of blood. (Trans. of Path. Soc. vol. xviii. 1867.)

Occasionally, however, an actual rupture of one of the minute vessels may take place under the increased strain upon its walls in cerebral congestion. This is all the more likely to occur in elderly people whose vessels have been weakened by fibroid or atheromatous degenerations. In such cases I have not unfrequently found, after careful preparation, evidences of past capillary hæmorrhages on several of the smallest vessels of the same brain. After the brain substance has been washed away, and when the vessels are floated in water in a shallow dish, one or more little orange-coloured specks may be seen, even smaller than a pin's head. On examination with the microscope these are found to be accumulations of altered blood pigment in the form of amorphous canary-yellow coloured flakes, interspersed with distinct crystals of hæmatoidine, situated around one of the minute vessels, and distending its sheath in a more or less obtuse fusiform manner. In these cases the presence of the perivascular sheath seems to have limited the amount of blood effused. As soon as the sheath became distended in the immediate neighbourhood of the rupture, the pressure so produced would tend to close the aperture in the ruptured vessel.

Lastly, there is to be mentioned that condition of certain parts of the brain which was spoken of by Durand-Fardel[1] as "l'état criblé," and which he and others regard as an evidence of previous dilatation of the vessels from long-continued congestions. This condition is occasionally well seen, more especially in old people, in the white substance immediately beneath the grey matter of the convolutions. On section a number of round or oval apertures appear—some large enough to admit a pin's head—and within each may be seen the cut extremity of a vessel. In these situations the canals in the nerve substance have become enlarged by pressure, and the lymphatic sheaths have been dilated to a similar extent, whilst in the space between the sheath and the much smaller blood-vessel a large quantity of pigment granules is generally met with. This dilatation of the vascular canals sometimes reaches an extreme degree in the corpora striata and in the optic thalami, and the same condition may be encountered, though to a less extent and less frequently, in the substance of the pons Varolii. Whenever the granules are met with, however, the structural conditions and the mode of origin seem to be the same. Durand-Fardel says:—"Tantôt l'état criblé du cerveau se trouve répandu dans une grande étendue des hémisphères, tantôt on ne l'observe que dans un espace circonscrit." This condition may be met with at all ages, though it is found more particularly in old people; and in them, the same writer tells us, the canals are sometimes so large and numerous in the corpora striata, that these bodies may seem to have lost nearly half their substance. It seems most probable that these canals have been produced by the dilatation and pressure exercised by congested vessels, though their method of pathogenesis cannot be said to have been ascertained in a thoroughly satisfactory manner.

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[1] Loc. cit. p. 57.

PROGNOSIS.—In cases of cerebral congestion regard must be paid to the age of the patient, the form of his attack, the severity of the symptoms, and the frequency with which the symptoms or the attack of symptoms may have occurred.

Age cannot be fairly estimated by the mere duration of life, for some men are "older" at fifty-five than others are at seventy years, and are so without any necessary co-existence of exhausting or definite disease. The apparent age is a truer guide than the real age in the matter of prognosis. Baldness, grey hair, rigid vessels, a weakened heart, arcus senilis, and enfeebled powers, must be taken into more serious account than the date of birth; and judged by such tests, the prognosis is unfavourable in proportion to the oldness or agedness of the individual.

The form of attack is worthy of most grave consideration. That which is of the worst omen is the apoplectic; next to this is that characterised by delirium; after it the convulsive form; and least serious of all is the febrile, or quasi-febrile.

The severity of symptoms is of much value in relation to the apoplectic form; the danger being in direct proportion to the profundity of coma, and its duration. It is of but little moment when delirium is the most prominent symptom, and the value that it possesses is in inverse rather than direct ratio to the force of the disturbance; the prognosis is worse when the delirium is mild, muttering, and continuous, than when it is noisy, or even violent, and—as is usually the case—of short duration. When convulsions occur, it is not safe to base any prognosis on the mere fact of their severity; for often patients recover after the most frightful seizures, whereas others succumb to much milder paroxysms. The degree to which, in the intervals of seizure, the mind is restored to its normal state, is a fairer criterion of the amount of danger than is the violence of the convulsion. In the febrile form the prognosis is bad in direct proportion to the intensity of the symptoms. When the disturbance is slight, confident hopes of recovery may be entertained; when it is severe, there is room for the apprehension of ulterior and "inflammatory" changes.

Congestion of the Brain is rarely fatal at its first attack; it becomes dangerous in proportion to the frequency and readiness of its induction; and this is true with regard to each form in which the symptoms may be developed.

The other conditions by which the prognosis must be determined are those of organic disease or degeneration in any of the important vital organs. It is obvious that the heart, the vessels, the kidneys, and the liver should be examined with care, and that the opinion formed as to the future should be guided by the kind and amount of disease that may be found in them. The prognosis, however, when such diseases are discovered, is not that of cerebral congestion only, but of those complicated morbid conditions of which it is but one form of expression. It may be that Congestion of the Brain is likely to prove the cause of death, but the nature of the disease which leads to such congestion furnishes the material, by a consideration of which the probabilities may be estimated.

TREATMENT.—As there are two distinct, practically opposite, conditions of the body under which cerebral congestion may occur, so there are two different lines of treatment to be adopted. If the brain congestion be but one of many symptoms of a general plethora, much may be gained by either general or local blood-letting; if it be but the outcome of weakness and vascular obstruction, then such measures may increase the evil. The previous habits and health of the patient, the present state of his integuments,—their warmth, vascularity, and colour,—the state of the pulse, of the heart and vessels, will furnish the guides in this important matter. A man in middle age, who has overstrained himself, or placed his head in some dependent position, and who is attacked by violent convulsions, characterised by great turgescence of the skin, bloodshot eyes, and a full but laboured pulse, may be relieved, and greatly relieved, by venesection to the amount of six or ten ounces. But such cases occur rarely, and in the majority of instances no man would at the present day think of bleeding from the arm. When, however, there is distinct general weakness, and, with this, heat of head, oppression, continuous headache, and a tendency to drowsiness, much relief may be obtained by the application of leeches to the temple, or by cupping to three or four ounces at the back of the neck. When there is no such heat of head, and no flushing of the face, but when the diagnosis of cerebral congestion may still be made—per viam exclusionis—and when the vital powers are low, the pulse small, feeble, irregular, or intermittent, even a small abstraction of blood locally may be followed by the worst results. It is when attacks of congestion are frequently repeated, and other measures have failed to relieve them, that local depletion may be found of signal service.

It is well to raise the head, to apply cold water or ice to the forehead, and to place the feet and hands in hot baths. If the stomach be overloaded, an emetic of mustard or ipecacuanha may be given; and often with the discharge of the stomach the symptoms pass away. This is especially useful when the attack has followed a full but hastily taken meal. It is of great importance to empty the rectum, and the most efficient means for doing this is the administration of an injection of warm water. Should there be any suspicion of the existence of hardened masses of fæces, the injection of a large quantity of warm olive oil will prove more useful than that of water.

When the tendency to cerebral congestion is noted, rather than any marked symptoms of its presence to a high degree, the secretions must be carefully regulated; and among these one of the most important is the urinary. Many cases of threatening aspect are to be relieved by saline diuretics; and I have known a copious flow of urine to be followed by the removal of symptoms which had existed in spite of free purgation and other treatment.

There are many cases occurring in advanced life in which the congestion is of only momentary duration; and the patient, when seen by the physician, is simply bewildered, pale, and with a cool, moist skin, and feeble pulse. Under such circumstances the cautious administration of stimulants is called for; and of these salvolatile and wine are the most useful. It is well to combine with them carbonate of potash, or of soda, as there is often considerable "acidity of stomach," and the discharge of flatus by the mouth, which results from such administration, is often followed by a complete remission of the symptoms.

As precautionary measures, quiet of mind, and gentle exercise of body, with the careful avoidance of either fatigue, sudden change of posture, or strain, should be enjoined; and much relief may be obtained by insuring a position during sleep which shall prevent not only the head, but the head and shoulders, from sinking down to the level of the body. This may be easily obtained by a simple contrivance placed under the bed or mattress upon which the patient lies; such an arrangement being much better than a mass of pillows, which shift their places, and often maintain the head in a condition of undue heat.

CEREBRITIS.

By J. Russell Reynolds, M.D., F.R.S.,[1] and
H. Charlton Bastian, M.D., F.R.S.

It is probable that general inflammation of the brain never exists alone, but that it is invariably associated with meningitis. The terms encephalitis, meningo-cerebritis, and phrenitis, which have been employed to denote the condition now referred to, are sufficient of themselves to point out this constant association. Nevertheless, in some cases there is to be found, during life, the predominance of a class of symptoms which simple meningitis will not account for; and, after death, the presence of such changes in the cerebral tissue, as do not necessarily accompany the meningeal inflammation. It would seem therefore, that the brain substance is not only susceptible of morbid change of an inflammatory type, but that the presence of such change may determine the clinical history of the case. We may, in particular instances, refer some of the symptoms of a complex encephalitis to inflammation of the membranes, and others to an implication, in like change, of the cerebral tissue.

Meningitis has already been described, and it remains for us, in this place, to describe only those symptoms which mark the extension of the malady to the brain itself. All that relates to that which has been described as local Cerebritis, or limited softening of the brain, will be found under the articles on Abscess of the Brain and Softening of the Brain.

Causes.—The most common causes are injuries to the head; such as violent contusions, wounds, diseases of the bone, and insolation. It would appear, however, that sometimes prolonged mental exertion or moral excitement have led to the development of this disease. In rare cases there has been no distinctly recognisable cause, the symptoms having appeared in the absence of any one of the conditions above mentioned.

Symptoms.—These are, of necessity, associated with those of meningitis, but sometimes they are the earliest to appear, and are predominant throughout the case. Thus, some mental change may be the first evidence of disease; it may be very slight, and may be mistaken for "hysteria," "stomach disturbance," or some such vague malady. In one case, which I saw several years ago, there was a mere confusion of ideas, and a worried manner, with misuse of words, and this for two or three days before other phenomena appeared. Usually the patient is sullen, and the faculties are obscured; there is a confused, "muddled" state of the intellect, sometimes merging into mild delirium, sometimes, when meningitis is present, alternating with, or superseded by, violent excitement.

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[1] The section on Pathology is written by Dr. Bastian.

There is deep-seated, oppressive pain in the head, described as sometimes shooting from the centre to the vertex, the temples, eyes, or ears; and this pain is persistent, and is out of all proportion to the pyrexia, which is often very slight. Except in dependence upon meningitis, there is no intolerance of light or sound, but there may be obscurity of vision, diplopia, and failure of sight, together with ringing noises in the ears, and some difficulty in hearing.

There is general muscular lassitude, but neither definite paralysis nor spasm; the limbs are weak and aching, but they may all be moved.

Such symptoms may continue for two, three, or four days, and then a violent convulsion may occur, followed by coma, from which the patient never thoroughly recovers. There is, however, partial recovery sometimes, and then more or less general paralysis is discovered. The patient is stupid, sleepy, comatose, and lingers for a shorter or longer time, in proportion to the amount of nourishment that can be given and retained by either stomach or rectum. Convulsions, somewhat epileptic in character, usually recur, and in their intervals there is to be observed a gradual dying out of the various functions of the brain. Mind, sensation, and voluntary power are lost, and the patient lives a mere vegetative life, disturbed occasionally by slight spasmodic movements, or rigid contraction of the muscles. The convulsions are often of long duration, involve the limbs especially, and are not marked by notable asphyxia.

The general symptoms are, as a rule, so slight that they attract no notice. There is no fever, little or no vomiting, and no obstinate constipation of the bowels. Very often the sphincters are relaxed quite early in the history of the case, and nothing abnormal can be discovered in the evacuations.

DIAGNOSIS.—That which gives to the diagnosis of meningitis its gravest element is the recognition of co-existing Cerebritis, and hence the diagnosis is valuable as an aid to prognosis. Cerebritis may be inferred when there is a rapid transition from the excitement of meningeal inflammation to the marked loss of function which is characteristic of cerebral change. When the signs of meningitis are unusually severe, the pain deep-seated, and followed after twelve or twenty-four hours by convulsions, coma, and paralysis, there is commonly Cerebritis of considerable extent.

PATHOLOGY.—Of uncomplicated Cerebritis we have no knowledge. When inflammation of the brain substance exists, it is either associated with a more marked change of the same kind in other parts, such as the meninges, in which case it is treated of as a concomitant condition, and not as a primary morbid affection; or else it speedily lapses into other distinct pathological states, such as abscess or softening, which, on account of their importance, are usually described as independent affections of the brain.

Two kinds of Cerebritis are usually described, namely, the diffuse or general form, and local Cerebritis, which by most recent writers has been held to be synonymous with "red softening," or "acute ramollissement" of the brain.

The diffuse form, or general Cerebritis, is a more or less wide-spread affection of the cortical substance, or grey matter of the convolutions, and is always associated with inflammation of the meninges. It may be met with in surgical cases, from injury to the skull; when, conjoined with it, there is inflammation of the dura mater and arachnoid, together with the formation of purulent lymph within the arachnoid cavity, and also beneath the visceral layer, into the meshes of the pia mater. Cerebritis may also be met with in the more limited meningitis, such as occurs when the disease is not of traumatic origin, and which, affecting the pia mater principally, is not accompanied by any purulent effusion in the sac of the arachnoid. In these cases there is extreme vascularity of the cortical grey substance, which is also more soft and pulpy than natural; and it is frequently adherent to the meninges, so as to be torn when these are stripped off. For further particulars we must refer to the articles "Meningitis" and "Tubercular Meningitis," under which heads these morbid conditions are more fully described. It should be stated, however, that many pathologists of the French school look upon general paralysis of the insane as a disease due in part to a species of chronic Cerebritis. The same adhesion between the grey matter and meninges is frequently met with in this disease; but for further information we must refer to the article on this subject.

With regard to local Cerebritis, we think with Lebert and other pathologists that this may be the antecedent condition and proximate cause of abscess in the brain; and we do not deny, also, that some acute softening of the brain may have an inflammatory origin. We do, however, strongly object to the view that all "red softenings," or "acute ramollissements," have to acknowledge this method of pathogenesis. We believe that most of the softenings hitherto placed in this category have been brought about by embolism or thrombosis, owing to the interference with the cerebral circulation thus induced; and that the characters usually considered as diagnostic of their inflammatory nature are capable of receiving a totally different interpretation, as may be seen on reference to the article "Softening of the Brain." In this view we are supported by many recent writers on the subject. With regard to the occasional existence of softening of the brain of inflammatory origin, we do not altogether disbelieve in its occurrence, only we plead ignorance as to the characters by which such softenings are to be distinguished from others of a degenerative nature, due to arterial or venous obstruction. We certainly think it is a pathological condition which occurs very much more rarely than the statements of some pathologists would lead us to imagine. It may, perhaps, be looked for most confidently in cases of wounds or injuries to the brain, or around adventitious products, as centres of irritation.

Prognosis.—The prognosis is as bad as it is possible to be. There is no probability of recovery when symptoms such as those above described have been developed.

Treatment.—Only palliative measures can be used with any advantage. We have never seen any good result from mercury given by the mouth or by inunction; nor from blisters, cupping, or other modes of blood-letting. Pain may be relieved by the application of ice; and spasmodic movements may be limited by sedatives, such as belladonna and Indian hemp; but beyond such relief of symptoms therapeutic art has failed.

SOFTENING OF THE BRAIN.

By J. Russell Reynolds, M.D., F.R.S., and
H. Charlton Bastian, M.D., F.R.S.[1]

Definition.—A disease characterised during life by impairment of mind, sensibility, and motility, and after death by diminished consistence and degeneration of the cerebral substance.

The disease now to be described is that which has been known as white or non-inflammatory softening; ramollissement blanc, or ramollissement non-inflammatoire.

Causes.—There is little that is satisfactory which can be said with regard to the remote etiology of Softening of the Brain. Among the conditions which predispose to its occurrence the most important is age, or agedness. Softening of the Brain is essentially a manifestation of decay, and this may be either the natural result of the wear and tear of a long life's work, or it may be the early outcome of excessive strain. The real cause is that waste of tissue which is unbalanced by repair, and this may come from the long continuance of work,—old age,—or the unhealthy severity of work, and its undue relation to rest. The proximate causes may be resolved,—as will appear in the section on "pathology,"—into morbid conditions of the vascular system. Neither sex, constitution, nor season of the year have been shown to exert any marked predisposing influence, nor has any distinct relation been made out between any one particular condition of the heart and cerebral softening. Degeneration of the kidneys and impaired nutrition of the heart and vessels are among the conditions which frequently accompany ramollissement; but these ought to be regarded as certain parts of a general change of which the cerebral softening is but another or a counterpart, rather than as predisposing causes of its existence. Vegetations on the valves of the heart may become detached and may block up one of the cerebral arteries; and thus their presence on the valves might be regarded as predisposing to Softening of the Brain. But it must be remembered that such vegetations when *in situ*, i.e. undetached, do not specially predispose to Softening of the Brain, that they may lodge in other vessels than those of the cerebrum, and that when they are carried from the heart to the cerebral arteries they become determining and not predisposing causes. If we may employ the term "predisposing cause" under these circumstances, we should do so to the general or constitutional state that has led to the production of vegetations rather than to the vegetations themselves.

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[1] The sections on Pathology and Pathological Anatomy are written by Dr. H. C. Bastian.

There is nothing definite to be said with regard to exciting causes. Attacks have sometimes followed violent mental or moral excitement, anger, abuse of alcohol, over-fatigue, or local injuries; but in the majority of cases no such conditions have been present, and in very many there has been a singular immunity from all apparent causes of disturbance. Exposure to cold has been followed by an apoplectic seizure, and one of the more frequent determining causes of an attack has been too free a purgation of the bowels.

SYMPTOMS.—Softening of the Brain may occur as either an acute or a chronic disease. It will be well, therefore, to describe the affection under two general headings, and first:—

ACUTE SOFTENING OF THE BRAIN.

1. *Premonitory symptoms.*—These may be absent altogether, but such complete immunity is rare; for Softening of the Brain most frequently occurs in those whose health has been for some time below the average, and very frequently in others who are the subjects of some distinct chronic and exhausting diseases. There is nothing so special in the character of the general condition which may precede Softening of the Brain as to render it of much value in the forecast of a patient's chances. There is often an enfeebled condition, with impaired nutritive power, shown in the general bearing of the patient, and more distinctly in the weakness of cardiac impulse, rigidity of arterial vessels, and local inequalities of temperature. These facts may be noticed for months or even years, but there is nothing in them that points specially to the brain as the organ which is likely to give way. In combination with symptoms of cerebral failure they are, however, of great significance.

The symptoms which, when thus combined, are premonitory of softening, are often those already described as characteristic of impending or actually developed congestion of the brain. (See p. 426, article "Congestion of the Brain.") They are,—headache, more or less constant in duration, and usually "dull" in character, dulness of sight or hearing, numbness, obscure pain, weight, or an indescribable sensation of "something wrong" in the extremities, slight confusion of thought, sleepiness, weakness of purpose, hesitation in judgment, irritability of temper, diminished control of emotion, deficiency of muscular power, a stooping gait, and tendency to cramp in the limbs. Sometimes the face assumes a dull, expressionless aspect when the patient is at rest, and he may pass hours in a state of apparent indifference to all around him; but when called upon to exert himself is able to resume his habitual manner, and do his accustomed work, although with some heaviness of manner and apparent effort. There may be occasional and slight symptoms of faintness, the face becoming pale, and the limbs cool; and such occurrences are of much significance. It sometimes happens, moreover, that the altered sensations above described are noticed more on one side of the body than on the other; and the fact of this limited distribution is highly indicative of impending evil. There may be a little dragging of one leg, or only a tendency to lean to one side when either walking or sitting; and this, when constant in its locality, is of much graver meaning than is a much greater amount of weakness when variable in its seat.

Such symptoms may continue for a shorter or longer time, and may precede either acute or chronic softening, and there is nothing in their nature, when existing only to the degree described, and which can alone be regarded as "premonitory," that furnishes any clue to the form which the developed symptoms are likely to assume.

2. *The developed symptoms* may occur in one of three distinct forms. The patient may either have an *apoplectic* seizure, be taken with *convulsions,* or may pass into a state of *delirium;* and it will be convenient to describe these forms separately, premising that sometimes they pass into one another, and that occasionally mixed cases are observed, in which stupor, delirium, and convulsions alternate.

(*a*) *The apoplectic form* may be very gradual or very sudden in its onset. When the former, there is an increase of the premonitory symptoms for days or weeks; when the latter, there may have been no special premonition, but the patient suddenly falls down in what is termed an "apoplectic fit," and he is said to have had "a fit," or "a stroke."

Very commonly the attack occurs after too long an abstinence from food, or when the patient is fatigued by too long a walk, or too protracted an effort; sometimes when, as in congestion of the brain (see page 426), he is making an excessive exertion. It is not common, so far as my experience extends, for patients to wake up in the morning and find themselves paralysed on one side of the body, a mode of attack by no means uncommon in the case of cerebral hæmorrhage.

The condition of the *mind* is highly significant. Transient excitement, talkativeness, irritability, or wandering of thought, amounting sometimes to mild delirium, may occur for a few minutes. The patient says or does something quite out of relation to his surrounding circumstances or previous conversation; speaks as if to some person he may not have seen for years, asks a question which refers to events long since passed, or in some other manner shows that he is "not quite himself;" is bewildered and "queer," vexed or pathetic; he makes some effort to get up and do some extraordinary thing which no one can understand, is impatient of attempts at dissuasion or control, looks faint, and becomes more or less insensible, sometimes falling to the ground, sometimes voluntarily sitting or lying down, as if merely fatigued, or disgusted with the stupidity of those around him, who do not understand what it is he wants to do.

The patient may for a few minutes be completely insensible, and when he is so, it is probably due to sudden congestion of brain, or to equally sudden anæmia of brain, either of which may be recovered from in a few seconds or minutes. When, however, the physician sees the case, he rarely finds absolute loss of consciousness. The patient lies quietly, in apparently profound sleep, snoring, and taking no notice whatever of the questions that friends ask in anxious and beseeching tones; but if spoken to sharply, told to put out the tongue, open the eyes, give the hand, or do any other simple thing, he responds at once, usually makes an awkward failure, and then relapses into his former state; if asked a question, he makes some inarticulate or unintelligible sound in reply, and again falls back into his heavy sleep, sometimes muttering to himself, but more commonly snoring continuously, or occasionally interrupting the rhythm of his snore by a long-drawn sigh.

In such a state the patient may continue for hours, days, or even weeks. There is often sufficient intelligence remaining for him, when roused, to swallow food, to recognise friends, to make efforts to say something; but so much dulness of apprehension, and so much difficulty of expression, that the real life is "hidden," and it is impossible to know that we are on such common terms with it that we can understand its meaning.

In some cases there is after a longer or shorter period marked improvement, the faculty of articulation returns, and a certain amount of conversation is possible to and with those who will give pains to learn the language that is spoken. The names of common objects are forgotten, or are confounded with those of others; and this, sometimes with such constancy that friends may understand what is intended, sometimes with such thorough want of uniformity that the meaning is unintelligible. Notwithstanding this great obscurity of expression, it may be perfectly clear that the patient himself knows distinctly what he means, is aware that he is wrong in his use of words, is vexed at his blunders, and ingenious in contriving means to counteract or avoid them. For example, he may know so well the words which he wants, and which he supplies by others in ordinary conversation, as to write down lists of words, and point to one or more of them in order to make up his sentences, or correct erroneous expressions. (See page 443.)

Occasionally, after being even profoundly affected, the mental condition may undergo great improvement, and the patient, although not perfectly restored, be carried back again to the point described in the notice of premonitory symptoms. But far more commonly there is no real restoration; some confusion may clear away, the stupor may be essened: but when these improvements have occurred the mind is found to be dulled and incompetent, and in a state of gradually increasing deterioration; week by week, and month by month, the patient is further and further removed beyond the reach of intercourse, until the attempts to talk with him are given up so gradually by the friends that they are scarcely aware of the change, and so imperceptibly to the weakening mind of the patient that he takes no notice. In this way a sudden attack of softening may pass into what is termed "chronic softening," the patient becoming imbecile of mind, and powerless in body.

In a certain number of cases, the course is rapid, the apoplectiform attack is repeated, and at the end of two or three days there is profound coma, passing into the sleep of death; in a much smaller number, the symptoms are very transient, and the recovery may be complete. For example, a young lady æt. twenty-two, in her first attack of acute rheumatism, marked by considerable swelling and redness of knees, wrists, and ankles, and a recently developed systolic murmur at the base of the heart, received a visit from some friends, was excited in conversation, and had palpitation of the heart. A few minutes after her friends had left her she grew faint, looked pale, became unconscious, and remained so for two or three hours. At the end of that time she was confused, unable to utter any intelligible sentence, clipped her words, made some sounds that were quite inarticulate, and had marked right hemiplegia, the features being drawn to one side, the right arm being completely, the right leg incompletely, paralysed. At the end of three days speech was perfect; and at the end of a fortnight the hemiplegia had disappeared. The most rational interpretation of such case is, that an embolus blocked up the left middle cerebral artery, and led to impaired nutrition of the brain, which equals the first stage of softening; but that, owing to either the re-establishment of the circulation by the breaking up or removal of the embolus, or to the perfect establishment of the circulation in the collateral vessels, the nutrition was restored to its ordinary condition.

Sensibility is sometimes quite destroyed at the time of attack, and for some few minutes afterwards; but in the greater number of cases it is only dull or impaired, and subsequently changed. While the patient is lying apparently unconscious, or only half conscious, it is often obvious that some sensibility is present, for he moans, moves about uneasily, puts his hand to his head, and starts or draws away some one or other of his limbs if the skin be scratched or pinched. It is probable, from the frequency with which the hand is put to the head, that there is headache, or an uneasy sensation in the head; and a general feeling of distress, with, very frequently, some distressing sensation in one or more of the limbs. Often before the patient is able to speak, he is evidently uneasy in some of the extremities, and these are usually on one side of the body, and are paralysed; he looks inquiringly at them, or rubs them, moans at them, and cries out if they be either moved or touched. When sufficiently conscious to make himself intelligible, the patient often complains of coldness, or numbness, or "queer feelings" in the arm or leg of one side; of headache, or discomfort in the head not amounting to pain; of a bewildered feeling, and some vertigo. There is occasionally hyperæsthesia, and its occurrence in the limbs affected by a stroke of hemiplegia is thought to be highly characteristic of acute Softening of the Brain. It would appear, however, that, instead of true hyperæsthesia, there is a modified sensibility, such as that described above, and that it renders ordinary impressions painful. Sometimes these modifications present very curious features; the patient feels distinctly and painfully any impression on the skin, but is unable to refer it to its proper locality. For example, a pinch on the sole of the foot is referred to the inguinal region, while similar irritation above the knee may be felt in the shoulder, or side of the neck; and sometimes the sensation may be referred to the wrong side of the body.

In the majority of cases the sensibility of the limbs is, after an apoplectic attack of softening, speedily restored to its normal condition.

The special senses are, as a rule, unaffected except in the earliest stage of the attack, when all of them may be in abeyance. There is not rarely some complaint of tinnitus, and of muscæ, or of dulness in hearing, or mistiness of sight, but there is no one change which is characteristic of softening. The optic disc is often paler than natural. The vessels are extremely small, and either white or grey atrophy may be apparent. The outline of the disc is sometimes very sharply defined, and its shape distorted; but there may be integrity in the appearances presented by the eye when there are unequivocal indications of Softening of the Brain. There is often to be observed some marked peculiarity in the eyeballs and in the pupils, which being, however, illustrations of altered motility rather than of sensibility, will be described under the following heading:—

The symptoms due to changes in motility.—It has been often observed that the eyeballs are directed to one side, and that the head is turned in the same direction, so as to give the idea that the patient in making an effort to look at something by the side of him, and usually on the opposite side to that of the paralysis in limbs. If carefully examined it may sometimes be shown that the patient does not see at all, and that the retina is quite insensible to light; while in other cases the patient sees distinctly, and may, by an effort of the will, bring the eyeballs to the middle line, or even beyond it and to the opposite side. In one curious case, under my care in University College Hospital, this synergic condition of the eyeballs was observed for nearly a fortnight, at the end of which time the patient died. The patient was, when roused, sufficiently sensible to give a coherent account of himself, his sight and hearing were good, he could distinguish not only objects but colours, and seemed rather amused at being put through any examination on such points; yet while talking to me the eyeballs were constantly turned towards the right side, and so much so as to hide a considerable portion of each iris beneath the lids at the left inner and right outer angles. The patient often fell asleep, and began to snore while the students were standing round his bed; and what was very interesting to observe was this, that at the moment of doing so the eyeballs returned to the middle line. Upon touching him, or speaking to him so as to rouse him a little, the synergic movement again instantly appeared. Owing to the ease with which the transition from sleeping to waking could be effected in this case, the above observation was repeated many dozens of times, and always with a similar result. The eyeballs are usually, except at or soon after the occurrence of the attack, unaffected. It is the rare exception, and not the rule, to meet with strabismus even to a slight degree. There is nothing characteristic in the condition of the pupils; they may be found in almost every degree of either dilatation or contraction, and they are usually equal on the two sides in cases of acute softening. Their relation to light is determined by the general sensorial condition rather than by any special involvement of their own motor centres. It has been said that occasionally the pupils dilate upon exposure to light and contract upon its withdrawal, but there has been, I believe, a fallacy in such observations which it is very easy to correct. The facts as they have been witnessed by myself in many cases are these: that the patient is found asleep, or in a state of half-unconsciousness, with contracted pupils; a strong light is brought before him, or the eyelids are suddenly raised, and then immediately there is dilatation; left to himself, again the drowsiness comes on, and the pupils pass into the state of contraction. The pupils dilate because the patient is roused, not because they are exposed to light; the contraction and dilatation that have been observed have had no relation to light or darkness, but simply to the facts of sleeping and of waking. This I have shown again and again by gently raising the lids of such a patient, and exposing the contracted pupils to the light without arousing him; there is then no dilatation nor change of any kind: but if he be addressed loudly by name, or if his toe be pinched so that he is awakened, the pupils instantly dilate.

The features are sometimes quite symmetrical, both when at rest and when in motion; but commonly there is some deviation, noticed most distinctly in the lower part of the face at the angle of the mouth when the patient speaks or laughs; and sometimes it is so trifling that it may escape observation unless the patient smiles, or makes a forced effort to exhibit either the upper or the lower teeth. In other cases there is marked paralysis of the face on one side, and dragging of the features towards the other, with deviation of the tongue; but let it be remembered that this paralysis does not, as a rule to which there are very few exceptions, involve the muscles of the forehead, eyeballs, eyelids, or pupils. The patients can equally raise the eyelids, open or close the eyes, and there is neither ptosis nor strabismus.

The speech is commonly interfered with, not only at the moment of attack but for a long time afterwards, and sometimes persistently. It may be so thoroughly abolished that no intelligible sound is uttered, although it is obvious that ideas of some kind are passing through the patient's mind; it may, on the other hand, be so slightly affected that alteration is observed only in the articulation of certain sounds, such as those of the letters *l* or *r*. Between these two extremes there is almost every variety of degree in the impairment of speech as a mechanical act, and there is also every shade of difference in the precision with which it expresses mental processes. Some patients can read with ease and correctness, articulating every sound distinctly; and yet they cannot construct for themselves a sentence of half a dozen words, so as to answer intelligibly the simplest question. Such patients, although able to hold a pen and to copy sentences, or sometimes to write a few words from dictation, cannot compose anything for themselves. In such instances language is interfered with on its intellectual side. Other patients can write well, when not flurried, can talk for a little time so as to be understood, can help to convey their meaning by signs and gestures; but when "excited," or sometimes even when not disturbed in such manner, they can make no such succession of articulate sounds as shall be intelligible. Here speech is interfered with on its mechanical side. In the former group of cases there is usually paralysis on the right side of the body: in the latter there is not any constancy in such association. It is to the former class that the terms "aphasia" and "aphemia" have been applied; and it is not rare to meet with cases which illustrate either it or the opposite condition: it is exceedingly easy to recognise intellectually the difference between the two extremes of symptoms, or between them as conjoint elements in a particular case; but by far the most common event is to meet with such combinations of the two that it is by no means so easy to say how much is due to the one failure and how much to the other.

As the words aphasia and aphemia have now passed into frequent use, and the conditions described by them have become not unfrequently the topics for medico-legal investigation, it is desirable that some further attention should be directed to them, or to what they mean. Aphemia was the word constructed by M. Broca;[1] and aphasia, an old Greek word, signifying the dumbness occasioned by strong emotion, was that used by M. Trousseau[2] to denote the same thing, viz. the loss of speech or of articulate language, when occurring as the symptom of disease.

The condition now well known as aphasia was observed by the older writers on medicine, some of whom appear to have recognised the distinction, and others to have failed to do so, between it and a more general condition of injury to the nervous centres. But the special pathological significance of the loss of language has been demonstrated within a recent period. Dr. Gall was the first who sought to

[1] Sur le Siége de la Faculté du Langage articulé: Bullet. de la Soc. Anat. 1861.

[2] Gaz. des Hôpitaux, 1864.

discover the locality or seat of what he, in accordance with a certain school of philosophy, was led to regard as the separate faculty of language, and he arrived at the conclusion that this faculty had its place in those portions of the anterior lobes of the brain which lie upon the supra-orbital plates.

The idea of Dr. Gall was taken up and strongly advocated by M. Bouillaud,[1] who distinguished, with care, between the recollection of words and the power of producing distinct sounds for their expression. M. Bouillaud's great point, however, was to show that lesions of the anterior lobes of the brain occasioned loss of the faculty of speech, whereas diseases of other portions of the nervous centres were not so accompanied. Exceptions to Bouillaud's law were somewhat frequently pointed out both in this country and in France, and until a few years ago the general doctrine had fallen into almost complete disregard, although M. Bouillaud had repeatedly brought forward fresh facts in confirmation of his dogma.

The next step of great importance was taken by M. Dax, who, in 1836, pointed out, as the result of his analysis of numerous observations, that disturbances in the faculty of speech were always related to lesions of the left hemisphere, and never to those of the right. This work, to which my attention has been called by the able author of the article on Aphasia, in the new "Dictionnaire encyclopédique des Sciences médicales,' was entitled "Lésions de la Moitié gauche de l'Encéphale coincidant avec l'Oubli des Signes de la Pensée."[2] The paper of M. Dax appears to have attracted little notice, and it was not until nearly thirty years had elapsed that M. Broca produced his celebrated paper,[3] in which he announced his conclusion that the seat of the faculty of articulate language was in the second, and especially in the third, frontal convolution of the left anterior lobe of the brain. M. Broca used the word aphemia to denote the condition of patients thus affected; M. Trousseau the word aphasia. The last step in this history, and one almost equal in importance to either that had preceded it, was taken by Dr. Hughlings Jackson, who arrived independently at a conclusion similar to that of MM. Dax and Broca, but who went still further than either, and showed the anatomical nature of the lesion which most frequently caused aphasia, viz. plugging of the middle cerebral artery on the left side by an embolus derived from valvular disease of the heart.[4]

Aphasia may be produced by numerous diseases of the brain—such, for example, as congestion, hæmorrhage, or tumour—but the most frequent cause is that to which Dr. Jackson directed especial attention. Certain aphasic patients can write, while others fail to do so ; those who are capable of the act occasionally write sense, frequently nonsense, but more frequently either unintelligible characters or distinct but unconnected words. Those who cannot write at all are usually, it must be remembered, paralysed on the right side. Certain patients are able to make intelligible signs, others fail to do so; some have the power to calculate, to draw figures, and to perform on musical instruments, while others lose these faculties together with that of articulate language.

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[1] Traité de l'Encéphalite.

[2] Gaz. Hebdomad. 28 Avril, 1865.

[3] Sur le Siége de la Faculté du Langage articulé, ant. cit.

[4] Loss of Speech, its Association with Valvular Disease of the Heart, &c. &c. : Clinical Lectures and Reports, London Hospital, vol. i. p. 388.

The mental state of the patient varies greatly, from a condition of almost perfect intelligence to that of almost complete fatuity. On several occasions the question has been raised whether an individual in a condition of aphasia should be regarded as competent to make a will. No general principle can be laid down with regard to so complicated a question, but each case must be determined on its own merits; still it must be remembered,—1st, That while intelligence may remain intact, the power of expression may be so damaged that it is impossible for any one to be certain that he has correctly interpreted the patient's meaning. 2d, That the patient may be distinctly capable of intellectual decision on matters up to a certain point of complexity, and quite incapable of dealing with those which are beyond that point; that he may be able to decide some simple question, whereas he becomes bewildered when attempting to unravel those which require sustained thought for their comprehension. 3rd, That the facts which are most valuable as bearing on this question are very rare, and contradictory, viz. those which are supplied by patients who have been aphasic, have recovered, have remembered their previous mental condition, and have been able to give an account of it. On the one hand, there has been obvious mental obscurity; on the other, perfect clearness of intelligence, although, as in the case of Lordat, the memory for words was lost as well as the faculty for their expression.[1] 4th, That the balance of evidence is to the effect that the mind is usually somewhat damaged, although its degree of impairment may vary between wide ranges; that the loss of speech in aphasia may co-exist with loss of mental power, but that it does not depend upon it. A patient may be dumb because he has no idea to convey, but such a person is not aphasic; he may have much to say, but be unable to find the words in which to express his thoughts,—such a patient is aphasic; he may be quite clear in his thought, quite certain of his words, and able to write them with facility, but he cannot speak articulately, because he cannot make the sounds he wants,—such patient is not aphasic in the true sense of the word, but is paralysed in either the tongue, lips, or palate, or in all of them together.

The condition of aphasia has its analogues in locomotor ataxy, writer's cramp, and allied affections; and similar physiological considerations will carry the explanation of these curious states to about the same level of precision. Talking, walking, and writing are each of them very complicated processes, and are, in man at least, the result of education. Artificial, as well as natural, associations of nerve-action are involved in each of them,[1] and the result which ought to come from such co-ordination may be stopped at any point. We cannot yet assert what is the primary loss in the condition of ataxy, or in that of writer's cramp, neither can we do so with any accuracy in regard of aphasia. In each of them desire, volition, and intelligence may co-exist with entire muscular capacity; and yet the patient who wishes, tries, and knows how to walk, to talk, or to write, and who, moreover, has full power in his legs, his arms, and his apparatus for articulation, is unable to accomplish his purpose: he staggers and falls in the one instance, he makes illegible scrawlings in the second, and meaningless sounds or unintelligible jumbles of words in the last.

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[1] Analyse de la Parole, &c., pour servir à l'Histoire de l'Alalie et de la Paralalie. Par le Prof. Lordat. Dictionnaire, ant. cit. p. 632.

The limbs are almost invariably paralysed when there is Softening of the Brain, sometimes on both sides of the body, but with far greater frequency on one side. The paralysis is usually not absolute; it is more marked in the upper than in the lower extremity; it is seen to its highest degree in the fingers or toes, to its lowest degree in the shoulder or hip, and with intermediate severity in the forearm and leg. There is commonly some spasmodic contraction of the muscles which are paralysed, and this may take the form of either tonic rigidity, or of occasional clonic, or even choreic movements. Sometimes the muscles are rigid at the very moment of the apoplectic seizure, but more commonly the reverse is observed—there is complete flaccidity—and not until after three or four hours is the stiffness of limb to be recognised. At first it is noticed only after repeated flexion and extension of a joint; subsequently it is persistent, and is not developed but simply exaggerated by attempts at movement. This rigidity is distributed with irregularity, and is noticed principally in the shoulder, elbow, and knee-joints; and in this respect it differs notably from what has been termed "late rigidity,"[2] viz. that stiffness of limb which comes on after paralysis has lasted for many weeks or months, and which is distributed like paralysis itself with prevailing frequency in the distal extremities of limbs, and undergoes a gradual diminution as the joints are tested, one by one, from below upwards.

The electric irritability of muscles I have often found to be absolutely normal, even when paralysis is complete, and it has remained so for a considerable time; its persistence or the reverse has not been determined by either the degree, duration, or locality of the paralysis, by the presence or absence of rigidity or of wasting. The irritability of the paralysed muscles, as tested by percussion, has been found often in considerable excess, and this when the electric contractility has been normal. Let it be distinctly understood that I am not now speaking of the force of muscular contraction, which is invariably, or almost invariably, diminished in a palsied limb, but of the readiness of response to electricity, which is tested—not by the vehemence of contraction in a muscle, but—by the weakness of the electric power which will bring such muscle into play. In this sense the palsied limb exhibits every condition of irritability, viz. the normal, excess, or diminution. It does not appear to be of any moment in the diagnosis of cerebral softening from any other lesion in the brain, but it is well to bear in mind the facts that have been mentioned, inasmuch as mistakes have sometimes arisen owing to a confusion of terms when the distinction is being made between cerebral and spinal lesion or disease.

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[1] See article "Writer's Cramp."

[2] See Dr. Todd's Clinical Lectures.

There may be no *general* symptoms in cases of acute softening; at the time of seizure the patient may have been in apparently good health. As already stated, however, this is not common. Usually the individual is "older than his years," as shown by aspect, manner, gait, premature baldness or greyness of head; he has been weak for some time, has been threatened with a "break-down somewhere;" has suffered from disease of heart or kidney; exhibits arcus senilis, and has a rigid pulse.[1]

Beyond these general states there is nothing to be noted; the patient may, and often does, vomit when the attack is beginning to clear away and some slight consciousness returns, but it is rare to meet with a repetition of the vomiting. The appetite often is quite good; the digestion, secretions, and evacuations, natural; the temperature, usually quite normal, is sometimes raised to a very high point, and in many cases is notably depressed in the affected limbs. When the temperature has been raised there has been considerable jactitation of the limbs, or convulsive movements, more or less general in their distribution.

(*b*) The *convulsive form* of acute Softening of the Brain is sometimes so distinct in its features that the case is more likely to be confounded with epilepsy than with hæmorrhage or congestive apoplexy. After a few of the premonitory symptoms already described (p. 447) the patient is taken in a fit, which passes through the ordinary phases of an epileptic attack; but the patient does not become profoundly stupid or sleepy afterwards. He is, perhaps, restless, and a little loquacious, or he may be simply quiet, but "not quite himself," for a few minutes, an hour, or even longer, when a second seizure follows, in its turn to be succeeded by a third, and so on; each convulsion being followed by increasing stupor, and almost invariably by paralysis.

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[1] In an interesting case recently under my care in University College Hospital, a man became suddenly hemiplegic and aphasic, and continued so until he died. There was distinct evidence of old valvular disease of the heart, the radial arteries were hard and visible, the brachial arteries when he bent his elbow stood out like large twisted cords, the pulsation of which was not only excessive and visible, but distinctly locomotive; there was highly-marked arcus senilis; the man was old, bald, and grey. The inference drawn from these symptoms was that there was softening in and below the left corpus striatum, and that the softening was due to disease of the cerebral vessels. On post-mortem examination the diagnosis was verified exactly as to the nature and locality or the disease; but, strange to say, the brachial arteries, which were inspected carefully, presented no unhealthy appearance, and exhibited their ordinary amount of elasticity when stretched between the fingers. The inference from this is important, viz.: that vessels may lose their elasticity during life, and that to such a degree as to form valuable guides in the diagnosis of disease, but yet there may be no atheromatous deposit, or other change in their physical appearance, which can be detected after death. The mode in which the function is performed during life is a better test of the physical capacity and condition of the organ than is the physical state of the dead artery when examined directly by the hand and by the eye.

Regarded closely, these facts are to be observed with respect to the history of the disease. The premonitory symptoms, although so slight as to have attracted little notice at the time of their occurrence, are found, when attention has been directed to them by the onset of convulsions, to have been highly significant of impending evil. They consist generally in some marked change in the mental condition of the patient, such as peculiar drowsiness, listlessness, weariness, impatience, or some flaw in memory, with distinct but momentary incapacity to understand what is said. There may be, however, some little hesitancy in speech, the mispronunciation of a few words, a little weakness of one side, or some vague feeling of *malaise*, with numbness of extremities, vertigo, or faintness, thought to be either hysteric or dyspeptic in their origin, until the fit occurs and demonstrates the gravity of their meaning. The convulsions, although resembling epileptic convulsions in the main, differ from them in certain particulars, viz. :—The tonic stage is but feebly marked, and there is not much asphyxia. Clonic spasms are more violent on one side of the body than on the other, and they continue for a long time. The patient does not "come out of the fits," but passes from one into another with no distinct intervening period of quiescence. The fits recur in increasingly rapid succession, and at length the patient can scarcely be said to be either "in the fit" or "out of it." He lies in a semi-comatose condition, occasionally muttering, and making movements which appear to be voluntary, but which are interrupted by spasmodic jerkings, by suspension of the breathing, or by momentary tetanic rigidity. Gradually the limbs on one side cease to exhibit voluntary movement—they fall heavily when allowed to do so; the eyeballs are often directed to one side, and the features lose their symmetry; and thus the case goes on until it passes from a convulsive into an "apoplectic" or paralytic form. The patient who was "taken in a fit," thought to be epileptic or dyspeptic, becomes distinctly hemiplegic, and the true nature of the case is recognised.

From this condition there may be partial restoration; after a few days of marked disturbance, intelligence returns up to a point varying through wide ranges, and the state of the patient may be that described in the previous section (see page 449). The convulsions, however, sooner or later, recur; either to be again recovered from, or to carry the patient beyond the reach of hope. The intervals between the attacks of convulsions may be either weeks or months; but in many cases the course is much more rapid, and the patient dies within twenty-four or forty-eight hours from the onset of the fits.

(*c*) The form marked by *delirium* has usually been preceded by distinct premonitory symptoms, and it is most commonly observed at advanced age. The patient suddenly "wanders" in his talk, becomes loquacious or restless, is busy in manner, exerts himself, seems tired, and falls asleep. He wakes up, somewhat confused, but appears "to be himself again" for a few days, or even weeks, when the confusion and delirium reappear, and are more persistent. There is no complete restoration, but gradually one side is found to be paralysed, or to be slightly weaker than the other. The delirium alternates with coma, more or less profound; and the patient passes into a state like that following either the apoplectic or the convulsive form.

The delirium, usually mild, is sometimes violent; but when it is so there is generally some distinct meningitis, and the case runs a rapid course, reaching its termination in a few days.

3. The *final symptoms* of softening, like the prodromata, are similar in their character whatever the form in which the attack takes place. The patient becomes more and more comatose, the paralysis extends to the sphincters, the respiration becomes embarrassed, and death follows, usually "without a struggle." Nothing is more gradual or more tranquil than the mode in which the sleep of such patients often deepens, almost imperceptibly, into the sleep of death.

A peculiar form of softening has been described by M. Duparcque, as occurring in children of precocious intelligence, the symptoms of this condition being the following:—headache with drowsiness, perfect integrity of the mind, exaltation of the special senses and of general sensibility, without fever, delirium, or convulsions. After death the only change which has been discovered has been Softening of the Brain. M. Duparcque denominates this disease "ramollissement blanc aigu essentiel chez les enfants."[1]

CHRONIC SOFTENING OF THE BRAIN.

The symptoms of this condition may follow an "apoplectic seizure," whether the latter has had for its anatomical basis congestion, hæmorrhage, or acute ramollissement. They may, on the other hand, be developed very slowly and insidiously, and may, or may not, be preceded by those phenomena already described as "premonitory" of acute softening.

There is diminution of intelligence. The patient is unable to pay attention, and consequently fails to receive new ideas. Subsequently memory is impaired, past ideas are not recalled with readiness, and there is general confusion and incoherence. Sometimes there is mild delirium, or merely a restless and excited manner, towards the evening of the day, or in the night; occasionally there is the monotonous repetition of a particular word or act, which may continue for hours, days, or even weeks.

In regard of emotion, the majority exhibit dulness, or some degree of melancholy; and it is not uncommon to find that the expression of feeling is very little under control, and the sufferer is said to have become "hysterical;" and this is often the earliest indication of failing power. (See article on "Hysteria.") In other cases laughing and crying are very common; but they occur without assignable cause, and without the apparent existence of any correspondent emotion. The intellectual weakness increases, and the patient becomes drowsy. At first he may be aroused, but subsequently there is profound coma, and the patient dies comatose. In rare cases, however, the intellect may be preserved throughout. The gradual failure, one by one, of the intellectual faculties is, *per se*, one of the most characteristic symptoms; and the peculiar monotony (of word or action) has led Durand-Fardel to a diagnosis in some obscure cases.

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[1] Archives générales de Médecine, Fév. 1852; quoted by M. Valleix, Guide de la Méd. prat., tom. 2me, p. 176.

The most common alteration of sensibility is cephalalgia. It exists in about half the cases, and is felt generally among the earlier symptoms; but sometimes it does not commence until an advanced period, and it generally disappears towards the close of life. Its intensity is highly variable, rarely so great as that of meningitis, or *à fortiori* of tumour; its locality is frontal in the majority, and it is not often confined to one side of the head. When pain is not present there is generally a sense of weight and confusion of head; and, as I have observed in many cases, such a peculiar sensation that the patient says he fears his "mind is going." Painful sensations are often present in the limbs; and they are sometimes referred to the surface, sometimes to the muscles, and in other cases to the articulations. These modifications assume the form of so-called hyperæsthesia, cutaneous and muscular; or of numbness, formication, &c. They are commonly limited in extent to the parts presenting motorial changes; and when this is the case, they are highly characteristic.

Diminution of sensibility is common, but anæsthesia is rare; and, in respect of the former, it usually exists in conjunction with paralysis. These changes are gradual and imperfect in their development; and it is uncommon to find complete anæsthesia of the special senses.

Unless an apoplectiform attack has taken place, the muscles rarely exhibit any sudden changes; but when such a seizure has occurred, there may be complete hemiplegia; the face, articulation, the tongue, and the limbs of one side being involved in paralysis.

Paralysis in the typical form of chronic softening is distributed generally, and developed gradually; weakness of the muscles preceding their complete removal from volitional control. Hemiplegia is the most common form that is observed when chronic softening has an abrupt commencement, but it is followed, in many cases, by general paralysis, incomplete in degree; and this is important as a distinction from the persistent paralysis of hæmorrhage. At first, one leg drags in walking, or one hand feels less strong than the other, and grasps less firmly. The diminution progresses in an intermittent course; complete paralysis lasting sometimes for a few minutes or hours, and then the power returning to a certain degree, and for a longer or shorter time; but general weakness is found outside the range of limited paralysis. The motorial changes may, however, be limited to particular groups of muscles; for example, those of the face, of speech, of one arm, &c. Spasm, of tonic character, exists with great frequency, and may be found in the paralysed or non-paralysed side, though much more commonly in the former. The rigidity increases gradually, and persists until within a few days of death, when it usually disappears altogether. Tremors or epileptoid convulsions may alternate with, or take the place of, tonic spasm; or there may be local clonic contractions, and the muscles may be unduly sensitive to percussion. General paralysis usually occurs for some days or hours before death; and then stertor, involuntary micturition, and universal flaccidity are present.

PATHOLOGY.—Since the year 1820 Softening of the Brain has received considerable attention from pathologists, and more especially from those of the French school. Previous to this time, it is true, such a pathological condition had been recognised and reported by several observers, and among them by Morgagni;[1] but their accounts are meagre and unimportant, so that for the first real description of the disease we have to refer to the works of Lallemand[2] and Rostan.[3] The first of these writers looked upon all softenings of the brain as of an inflammatory nature, and there can be little doubt that many of the cases he described were really instances of cerebritis of traumatic origin associated with inflammation of the meninges; whilst Rostan, whose observations were made upon people of an advanced age at the Salpêtrière, thought these affections were sometimes inflammatory, and sometimes not. The latter, also, first called attention to the fact of the frequent association of softenings of the brain with calcification of the arteries in old people. Since this time the opinions entertained by different writers as to the nature of softenings of the brain have been various, though for the most part they may be ranged under two principal categories, since the subject which has always been most in dispute (and which cannot now be said to be entirely settled) has been, whether we are to regard these affections as inflammatory or non-inflammatory in their origin. The great though pardonable error of the earlier pathologists was, that they looked upon Softening of the Brain as a single substantive disease, instead of regarding it, as we now do, as the pathological sequence of various more or less different conditions. And, as we shall hope to show, much of the difference of opinion amongst later pathologists has been engendered and propagated because they have looked at various kinds of Softening of the Brain too much from the mere point of view of morbid anatomy. Thus some have attached an undue importance to certain appearances the real nature and value of which could only be rightly estimated by a consideration of the pathology and mode of origin of the lesions in question.

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[1] De Sedibus et Caus. Morb. t. v., Epist. v. ix. lvii.
[2] Rech. Anat. Path. sur l'Enceph. 1re lettre, 1820.
[3] Rech. sur le Ramolliss. du Cerveau, 1820.

Before dwelling upon this point further, I will briefly indicate the nature of the principal fluctuations of opinion amongst successive writers on this subject, whose works have followed those of Lallemand and Rostan.

Cruveilhier[1] held that certain forms of softening were of an inflammatory nature, but that certain others were not, these latter being supposed to be more allied to softenings of the stomach and intestines. His "ramollissement apoplectique," or "apoplexie capillaire," as he afterwards termed it (answering to some of the forms of red softening), he did not regard as inflammatory in nature, but he thought that this condition passed by almost insensible gradations into one of ordinary apoplexy. Bouillaud[2] at first proclaimed the inflammatory nature of softenings of the brain, though afterwards[3] he acknowledged the difficulty of the question and the desirability of further investigation on the subject. Andral[4] rejected the inflammatory doctrine. He spoke of obliteration of arteries and poverty of the blood as probable causes, but he also regarded the state of softening as due to a special alteration of nutrition which might supervene under the influence of the most different conditions. Abercrombie[5] looked upon softening as a species of gangrene, but spoke of two forms, one of which was essentially inflammatory in its origin, whilst the other—principally met with in old people—was due to disease and obstruction of the cerebral arteries. Much the same views were entertained by Carswell[6] and Copland;[7] and others afterwards insisted, as Abercrombie had done, upon the importance of obliteration of the arteries in connexion with Softening of the Brain. Amongst the earlier of these may be mentioned Bright,[8] Crisp,[9] Piorry,[10] Gely,[11] Gueneau de Mussy,[12] and Bouchut.[13] But there appeared in France, almost at the same time, two of the most decided advocates of the inflammatory nature of Softening of the Brain, namely, Gluge,[14] who founded his theory upon the supposed nature of certain granular corpuscles or cells, to which we shall subsequently have to refer, and Durand-Fardel.[15] Those who had already called attention to arterial obstruction as a cause of cerebral softening referred to coagulations occurring in the vessels themselves, but Virchow, in his first memoir upon embolism,[1] opened up a fruitful and entirely new field for inquiries into the pathogeny of Softening of the Brain, which has since attracted the attention of many investigators, whose labours have yielded the most important results. Amongst others who have contributed to elucidate this aspect of the question, I may mention Kirkes,[2] Fritz,[3] Schutzenberger,[4] Oppolzer,[5] Cohn,[6] Lancereaux,[7] and Hughlings Jackson:[8] of special importance also are the experimental investigations of MM. Prevost and Cotard.[9] Reference may, moreover, be made to the work of Lancereaux for further historical information, and to the recent volumes of the "Transactions of the Pathological Society of London" for numerous cases recorded by English pathologists.

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[1] Introd. à l'Etude de la Médecine pratique (1821) 1er cahier, p. 112.—Anat. Patholog.—Dict. de Méd. et de Chir. prat., Art. "Apoplexie."

[2] Traité de l'Encéphalite, 1825.

[3] Dict. de Méd. et de Chir. prat. t. xv. p. 793.

[4] Précis d'Anat. Path. 1829, and Clinique Médicale, transl. by Spillan, 1836, p. 160.

[5] Path. and Prac. Research on Diseases of the Brain and Spinal Cord. 3d ed. 1836, p. 22.

[6] Path. Anat., Art. "Softening," and Cyclop. of Pract. Med. vol. iv.

[7] Dict. of Pract. Med.

[8] Guy's Hosp. Rep. No. 1.

[9] Lancet, 1840. Cases of Cerebral Disease.

[10] Bulletin Clinique.

[11] Gazette Médicale, 1838.

[12] Archiv. gén. de Méd. 1re Série, t. xxvi. p. 559.

[13] Actes de la Soc. des Hôpitaux, 1850, t. i. p. 43.

[14] Comptes Rendus, 1837, et Archiv. de Méd. Belges, 1840.

[15] Traité du Ramollissement, 1843, et Malad. des Vieillards. Paris, 1854.

In spite, however, of the light thrown upon the pathology of the disease by the recognition of the frequency of its association with embolism of the cerebral arteries, Durand-Fardel,[10] in 1854, again described the various softenings of the brain as inflammatory affections; and a few years later this view received the support of Calmeil.[11] Rokitansky [12]—as the representative of the Vienna school—also pronounced in favour of the inflammatory nature of red softenings; and amongst British pathologists there are some who still look upon various forms of Softening of the Brain as inflammatory, principally from the fact of the occurrence, in the softened tissue, of Gluge's granule cells, which are erroneously supposed to be produced only by a process of inflammation. The latest French writers, however, seem almost entirely agreed as to the non-inflammatory nature of the great majority of cerebral softenings, as may be seen by reference to the works of Lancereaux,[13] Laborde,[14] and Proust,[15] and to the memoirs of Bouchard,[16] and of MM. Prevost and Cotard.[17]

The acute course run by many softenings of the brain, and the red and swollen appearance of the part after death, appear at first sight to lend support to the doctrine of their inflammatory origin, though, after recent inquiries into the effects of obstruction to the circulation in different parts of the encephalon, it will be found that we shall have no difficulty in otherwise accounting for these phenomena. It seems impossible now, moreover, to look upon the large granule corpuscles, or so-called

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[1] Archiv für Anat. und Physiolog. 1847.

[2] Med. Chir. Transact. vol. xxxv. p. 281.

[3] Gazette Hebdom. 1857.

[4] Gaz. Méd. de Strasbourg, 1857, p. 50.

[5] Wien. Med. Wochensch. 1859 and 1860.

[6] Klinik der Embol. Gefasskrank. Berlin, 1860.

[7] De la Throb. et de l'Emb. Céréb. Paris, 1862.

[8] London Hosp. Reports, vol. i.

[9] Recherches phys. et path. sur le Ramolliss. cérébrale. Gaz. Méd. de Paris, 1866.

[10] Loc. cit.

[11] Malad. Inflammat. du Cerveau. Paris, 1859.

[12] Path. Anat. (Syd. Soc.) vol. iii. 1850.

[13] Loc. cit.

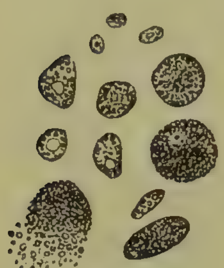
[14] Le Ramolliss. et la Congest. du Cerveau. Paris, 1866.

[15] Des diff. Formes de Ramolliss. du Cerveau. Paris, 1866.

[16] Archiv. génér. de Méd., Mars 1866.

[17] Loc. cit.

"compound inflammation globules" of Gluge, as products of inflammation only. These bodies are constantly present in all but the most recent patches of softening, and it seems to have been the tolerably wide acceptance of Gluge's opinion as to their origin, that has kept up the doctrine of the inflammatory nature of cerebral ramollissement. In their most typical form these bodies present themselves as large, spherical, or somewhat elongated aggregations of minute granules, generally about $\frac{1}{1000}''$ in diameter, though they may vary from $\frac{1}{0002}''$ to $\frac{1}{005}''$ in diameter. They may be surrounded by a delicate cell-wall, or this may be no longer visible. Occasionally a clear space, indicative of a nucleus, may be detected in their interior. From the fact that bodies in every way similar have been found by Turck,[1] Bouchard,[2] myself,[3] and others in secondary atrophic degenerations of the spinal cord, in which there is a simple process of wasting and not the slightest suspicion of the existence of an inflammatory process, there is the strongest evidence in favour of the opinion of Virchow, Robin, and other histologists, that these bodies result from the degeneration of pre-existing cells by the accumulation of fat and protein granules in their interior. They are, according to Virchow, produced from the cells of the neuroglia,[4] or connective tissue of the brain: and on this supposition we may easily account for the presence of granule corpuscles in the midst of the white substance of the brain, where formerly no cells were thought to exist. When these connective-tissue elements undergo the fatty and granular degeneration, they appear greatly to enlarge in size, the cell-wall becomes progressively thinner till it at last disappears, and ultimately the spherical aggregation breaks down into a mass of granular débris. It is thought also, by some, that granule corpuscles may originate in part by the aggregation of molecules originally separate, such as are always plentiful in tissues undergoing degeneration; and by others, that they may arise from the granular and fatty degeneration of the drops of myeline set free from the nerve fibres in softening nerve tissue. It must, however, be very difficult to substantiate either of these modes of origin.



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The extreme vascularity of the brain, and its naturally soft consistence, must be taken into consideration if we wish to understand how it is that diminution in the nutrition of any of its parts, and the degeneration which is its accompaniment, should lead to such marked alterations in consistence as are met with in cerebral softenings. Bearing these peculiarities in mind, however, the lowering of nutrition from vascular obstruction, with its consequent effusion of serum, together with the degeneration subsequently taking place, seems adequate to explain all the degrees of ramollissement which exist during life. Theoretically we should be compelled to admit that Softening of the Brain might be brought about by (1) an improper state of the nutritive fluid or blood; (2) by a want of due activity in the elements of the tissues themselves; and (3) by an impediment to the proper circulation of the blood. As matter of fact, however, we can say nothing positive concerning the first cause, as to whether it is capable alone of producing a condition of cerebral ramollissement. It can probably be looked upon only as a predisposing cause, under the influence of which softenings might occur, in cases where there was a concurrent action of even slight determining causes. Where the vitality of the tissues has been lowered in anæmic and cachectic states of the system, such as we meet with in patients suffering from cancer, we can easily imagine that this poverty of blood would be a powerful predisposing cause, though we must also take into account the fact that these states of the system also tend to produce some of those changes in the vessels which are so frequently instrumental in bringing about Softening of the Brain. We can say little, also, that is definite as regards the influence of the second cause, though a primary fatty degeneration of the nerve elements leading to one form of softening has been described by Dr. Hughes Bennett.[1] A proper activity of the elements of the tissues themselves is certainly one essential in healthy nutrition, and to its gradual failure we may perhaps attribute the occurrence of many of the pathological changes characteristic of old age. Doubtless, both alterations in quality of blood, and diminished nutritive activity of tissue elements, may be looked upon as accessory causes of no unfrequent occurrence in the production of cerebral softening, especially in old people. But undoubtedly the most frequent causes of Softening of the Brain, at all ages, are to be looked for under the third head, which includes all the varieties of impediment to the circulation of the blood. These may be classified in the following manner:—

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[1] Compt. Rend. Acad. des Sc. de Vienne, Mars, 1857.

[2] Archiv. gén. de Méd., 1866, p. 281.

[3] "On a Case of Concussion-lesion of the Spinal Cord, with extensive ascending and descending secondary degenerations." Med.-Chirurg. Trans. 1867.

[4] "Myelocytes" of Robin.

Morbid conditions of cerebral vessels	Obstructing circulation	Arteries	Embolism. Thrombosis.
		Capillaries	Embolism.
		Veins and Sinuses	Thrombosis.
	Preventing osmosis and nutritive exudation		Diseases of coats of capillaries and small arteries.

1. *Obstruction of Arteries.* *a. Embolism.*—The fibrinous masses of which emboli are composed have their origin for the most part in the left cavities of the heart and in the arch of the aorta, though more rarely they may proceed from the pulmonary veins. They may consist of portions of the fibrinous vegetations which are often met with on the mitral or aortic valves, as the result of endocarditis or atheroma, or of portions of those fibrinous depositions that are apt to form

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[1] Clinical Lectures. Fourth Ed. p. 355.

on the rough edges of atheromatous and calcareous patches of the arch of the aorta when these are not situated beyond the origin of the cerebral vessels; whilst at other times they may be constituted by detached portions of old clots which have been formed in the left auricle—or even in the left ventricle in cases where there has been a retardation of the force of the blood current, either owing to fatty degeneration alone or in combination with extreme dilatation of the heart. Virchow believes that cerebral emboli may proceed also from clots formed in the pulmonary veins; and it seems possible that small cancerous masses, swept away by the pulmonary veins in cases of carcinoma of the lungs, may occasionally go to form cerebral emboli, since in two instances small cancerous fragments have been found in the healthy heart and aorta—once by Lancereaux,[1] and once by Vidal.[2]

b. Thrombosis.—The various causes of thrombosis have been so well put by MM. Prevost and Cotard,[3] that we cannot do better than follow their arrangement.

(1) Pathological changes taking place in the walls of the arteries, by which their calibre is often much narrowed, and their lining membrane roughened, are conditions most favourable for the occurrence of thrombosis. Changes of this kind, whether atheromatous or other, directly favour local coagulation.

(2) Retardation of the rapidity of the circulation also predisposes to coagulation of the blood in situations where other conditions favourable to its occurrence are present. This retardation may be brought about by:—*a.* Diseases which weaken the force of the heart, such as dilatation without proportionate hypertrophy; and also, more especially, fatty degeneration of this organ. *b.* Narrowing of the calibre of arteries from atheromatous and other degenerations diminishes the rapidity of the circulation in these parts, and so predisposes to local coagulation, independently of the roughened surface with which the narrowing is usually associated. *c.* The loss of elasticity in the arterial walls, as a result of their degeneration, also assists in bringing about a retardation, since M. Marey has shown[4] that the elasticity of the arteries increases the rapidity of the blood current.

(3) And lastly, there are certain special states of the blood which seem to predispose towards the formation of arterial and venous thrombosis—sometimes so strongly as to bring this about even without an actual diseased condition of the vessels themselves. This tendency is most marked in the cachectic states of the system before alluded to, and it is said to exist more especially in those cases in which the cancerous diathesis is well marked. Here, however, it seems probable, that the feebly acting, and perhaps degenerated heart, may be almost as instrumental in bringing about the coagulation, as any special

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[1] Bulletin de la Soc. Anatomique, 1858.

[2] Comp. rendu de la Soc. de Biologie, 1861.

[3] Gaz. Méd. de Paris, Mai 19, 1866, p. 336.

[4] Physiolog. Méd. de la Circ. Paris, 1863.

alterations in the nature of the blood itself. There would be a conjoint action of these two predisposing causes.

All the conditions predisposing to thrombosis not unfrequently co-exist in many old people, and this fact harmonizes well with the extreme frequency with which softenings of the brain, not due to emboli, are also met with in the same subjects. Any of the cerebral arteries may become the seat of degeneration in old age, so that thrombosis, and softenings due to this cause, may be met with in the most various regions of the brain. But the middle cerebral arteries are those which seem more especially liable to embolic occlusion, and, according to some observers, that on the left side is more frequently occluded than its fellow on the right. There still seems to be some doubt, however, as regards this latter point. Thus, in forty-four cases collected by Lancereaux,[1] although the left internal carotid and its branches were occluded fourteen times, and the right only twice, the left middle cerebral artery was affected twelve times, and the right twelve times; some arteries of the pia mater near the left cerebral peduncle once; and the basilar artery and its branches three times. In eighteen cases reported by MM. Prevost and Cotard,[2] the same frequency of occlusion of the middle cerebral arteries was found, though the numbers are higher for the right than for the left side. Thus, the right middle cerebral was occluded seven times, and the left three times; the right anterior cerebral twice, and the left twice; the basilar artery once; the right internal carotid twice, and the left once.

It should be mentioned also, that in the experiments of MM. Prevost and Cotard, in which they injected tobacco seeds into the carotid arteries of dogs, it was almost always found that the Silvian or middle cerebral arteries were more especially occluded. In the statistics of thirty-two cases reported by Meissner,[3] the situations are found to be somewhat different. Thus, the most frequent seats of obliteration were ascertained to be at the termination of the carotids. The obstruction was met with in this situation seven times in one of these arteries only, and twice in both at the same time. Next in order of frequency stood the posterior cerebral artery, in which the obstruction was met with eight times; then came the Silvian artery, seven times; the basilar artery four times; whilst in the vertebral it occurred once in one artery, and once in both; and in the artery of the corpus callosum twice.

In almost every case where Softening of the Brain is associated with thrombosis or embolism of the cerebral arteries, it is found that the obliteration exists in one of the branches beyond the circle of Willis,[4] even though obliteration of the parent trunk also exists at some point before it gives off the branches for this anastomosis. Obliteration of the trunk of the carotid alone is not sufficient, under ordinary circumstances, to produce cerebral softening, as may be seen from a *résumé* by M. Ehrmann,[1] of cases in which the carotid arteries having been tied, the operation was followed by cerebral disturbance. The symptoms of cerebral mischief at first set up gradually disappeared when the circulation was re-established by means of the circle of Willis; and where softening did actually occur, this was due either to the extension of a clot upwards, beyond the circle, into one of the cerebral arteries, or perhaps, as M. Ehrmann suggests, to some unusual distribution of the arteries themselves at the base of the brain, preventing the establishment of a collateral circulation, such as ordinarily takes place.

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[1] Loc. cit. Mai 19, 1866, p. 337.

[2] Loc. cit. p. 19.

[3] Zur Lehre von der Thrombose und Embolie. Schmidt's Jahrbuch, 1861, No. 1, p. 89.

[4] On this subject Dr. Kirkes wrote:—"Although by the arrangement of the vessels composing the circle of Willis ample provision is made against obstruction ensuing in any of the main arterial channels on either side *previous* to their arrival at the circle, there is comparatively little provision for an obstruction ensuing in any of the main branches into which this arterial circle breaks up. This remark applies chiefly to the middle cerebral artery, which, if plugged at its origin, becomes at once altogether

The seat of the softening also corresponds with the anatomical distribution of the branch occluded, though the two are never coextensive. Usually the brain in the peripheral portions of the vascular department is healthy, owing to this portion of its tissue being nourished by the collateral capillary circulation, whilst the central portions of the vascular region are principally affected: thus, as Lancereaux points out, in cases of obliteration of the Silvian artery, softening of part of the corpus striatum and of the neighbouring white substance is generally observed, whilst the grey matter of the convolutions as well as the walls of the ventricle are often intact. It has been suggested by Durand-Fardel that the obliteration of the arteries is secondary to the softening, and not the cause of it: but, in reply to this, it is only necessary to state that the actual seat of arterial occlusion is almost always outside the softened tissue, and in these cases, as well as in those in which there is obliteration of the arteries within the softened patch itself, an examination of the vessels will either show degenerated and roughened walls together with the presence of an adherent clot within, or else it will establish the existence of a small obstructing mass, differing from recent fibrine in composition and appearance, and unattached to the walls of the vessels.

2. *Obstruction of Capillaries.*—In certain cases, by the rupture of old clots of the heart having softened centres, or by rupture of the inner coat of the aorta over large softened atheromatous patches, a mass of granular débris is carried into the cerebral arteries, whilst, from the minute size of the particles of which it is composed, these penetrate to and block up the minute arteries and capillaries of the part. If the quantity of matter thus carried to the brain be considerable and widely dispersed, death may rapidly follow before there is time for definite alterations of the cerebral tissue to take place, and owing to the extent of the capillary obliteration the brain, it is said, may present an anæmic appearance. Such was frequently found to

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useless as a blood-vessel, for nearly all its divisions, especially those for the central parts of the brain, proceed to their several destinations without receiving any anastomosing branch from the other divisions of the circle of Willis."—*Med.-Chir. Trans.* 1852.

[1] Thèses de Strasbourg, 1859.

be the case by MM. Prevost and Cotard, when they injected fine lycopodium powder into the carotid arteries of dogs. When a smaller number of capillaries are obliterated, either by atheromatous matter, by small particles of fibrine, or by pigment granules,[1] local patches of softening may be produced, having the usual characters of Softening of the Brain due to arterial obstruction.

3. *Obstruction of the Veins and Sinuses.*—The general causes favourable to the production of thrombosis have already been mentioned. The cases of obliteration of the cerebral veins and sinuses are in part due to some of these, though, just as frequently, they are the sequences of blows on the head, or of inflammatory conditions of the scalp and cranial bones. Indeed, out of the seventy-four instances of thrombosis in the cerebral sinuses which have been recorded by Lancereaux,[2] and other observers, such as Tonnelé,[3] Rilliet and Barthez,[4] Lebert,[5] Gerhard,[6] and Von Dusch,[7] thirty-nine are found to belong to this latter category. Amongst these, in no less than thirty cases it was due to caries of the bones of the skull: in so large a proportion as twenty-four of these cases it was the temporal bone that was affected as a result of otitis. Both the lateral sinuses are seldom implicated at the same time in these secondary thromboses, and the longitudinal sinuses are even more rarely affected from such a cause; whereas in those cases in which the thrombosis proceeds from more general causes, such as alterations in the quality of the blood or slowness of circulation, its almost habitual seat is found to be the superior longitudinal sinus, from which the thrombus frequently prolongs itself down to the torcular Herophili and then on each side into the lateral sinus. It is in this latter class of cases, moreover, that cerebral softenings are associated with the thrombosis. These are of a peculiar kind, consisting principally of a number of small patches of red softening, occupying chiefly the grey matter on the upper surface of the brain; and they are often distributed symmetrically over both hemispheres. Occasionally, softening of a portion of brain tissue of considerable extent has been noted. Besides such peculiarities in the seat and distribution of the softened patches, we usually meet, in these cases, with serous effusion into the ventricles and beneath the arachnoid, or more rarely with an actual effusion of blood in these situations or into the substance of the brain itself, together with many minute patches of hæmorrhage in the grey matter, such as have been described by Cruveilhier under the name of "apoplexie capillaire." The actual combination of these conditions met with in individual cases depends upon the seat of the obstruction, the rapidity with which it is brought about, and the condition of the vessels themselves. In the secondary thromboses, on the other hand, there is often evidence of

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[1] Lancereaux, loc. cit. p. 106; Frerichs, Traité des Maladies du Foie, p. 264; and Charcot, Gaz. Hebdom. 1857, p. 659.

[2] Loc. cit. p. 116.

[3] Journ. Hebd. de Méd. 1829, p. 337.

[4] Malad. des Enfants, t. i. p. 161, 1853.

[5] Virch. Archiv, Bd. ix. p 381.

[6] Deutsche Klinik, 1857, No. 45.

[7] New Syd. Soc. vol. xi. p. 81.

more or less circumscribed inflammation of the meninges, although the cerebral softenings and extravasations of blood very rarely occur. This, according to Von Dusch, is owing to the fact that in these cases the thrombosis starts from the veins in communication with the inflamed spot, and reaches the sinus only after the collateral circulation has had time to establish itself, instead of forming at once in the sinus, and before a collateral circulation has been set up.

4. *Alterations in the walls of the Capillaries.*—Fatty degeneration of the walls of the capillaries has been described by Hughes Bennett,[1] Paget,[2] Todd,[3] Moosherr,[4] and Charles Robin.[5] This alteration is most frequent in old age, and is said to be especially common in individuals suffering from Bright's disease, or from other maladies producing a low cachectic state of the system. In some of these cases such changes may supervene at a much earlier period than is usual. It is thought that such changes may not only favour the occurrence of cerebral hæmorrhage, but that they may also lead to softening when the changes are universal and well marked in the capillaries of a certain area. Such degenerations of the walls of the capillaries must not however be confounded with the accumulation of fat granules and of granule corpuscles on the walls of capillaries[6] which are situated in the midst of softened brain substance. The first state may possibly be a cause of softening, but the second condition is always a consequence of it.[7] The observations of Moosherr and Robin, more particularly, have shown that a certain number of fat particles may almost invariably be found within the sheaths of many of the small arteries and capillaries of the brain when this is quite healthy, and that, too, even in children. In many cases it is extremely difficult to discriminate between small fat particles and calcareous granules[8] in the walls of the capillaries, without submitting them to the action of dilute hydrochloric acid. This calcareous degeneration of the capillaries is more rare than the ordinary fatty degeneration, though when it exists in an extreme degree, it is also capable of giving rise to Softening of the Brain, as may be seen by the perusal of a remarkable case reported by M. Delacour,[9] in which the small arteries and capillaries were completely calcified. In these cases, as well as in those of fatty degeneration, the softening is brought about by a gradual diminution in the nutrition of a portion of the brain, the capillaries of which have been altered in structure so as no longer to permit the osmosis of a quantity of blood plasma sufficient to maintain the ordinary balance of nutrition in the surrounding tissue, and to prevent it from undergoing processes of degeneration.

[1] Edin. Med. and Surg. Journ. 1842.
[2] Medical Gaz. 1849, and Surg. Pathol. (revised by Turner) 1863, p. 106.
[3] Clinical Lectures on Paralysis, &c. 1854.
[4] Ueber das Patholog. Verhalt. der Klein. Hirngef. Wurzburg, 1854.
[5] Compt. rend. de la Soc. de Biolog. Paris, 1855, p. 142.
[6] Wedl, Patholog. Histol. (Syd. Soc.) p. 291, fig. 64.
[7] Billroth, Archiv der Heilkunde, Drit. Jahrgang. p. 47.
[8] Jenner, Med. Times and Gaz. January 31, 1862.
[9] Gaz. des Hôpitaux, 1850, p. 107; also Wilks, Journ. of Ment. Sc. vol. xi. p. 191.

In addition to the various softenings of the brain, which may be produced by the influence of some of the conditions already mentioned, and others of traumatic origin, which are mostly "red," owing to effusion and dissemination of blood, there are also *secondary* or consecutive forms of softening, which may be classified under two heads, viz.:—1. Softenings set up around tumours and adventitious products generally, in the brain. 2. Atrophic softening due to the separation of nerve fibres from their ganglionic communications. The first variety of secondary softenings will be referred to elsewhere (Art. "Adventitious Products"). Those coming under the second head are by no means frequent; the lesion resulting from the separation of a tract of nerve-fibres from their central ganglionic connexions being usually a simple atrophy or slow wasting. Although the method of degeneration, in this condition and in softening, has been proved to be identical, nevertheless actual cerebral softening does not usually occur, apparently because the atrophic change is brought about rather more slowly and without the occurrence of obstructions in the vessels of the part capable of producing œdema. Still, softenings from this cause have been met with. This kind of atrophic change was pointed out by Cruveilhier[1] in the cerebral peduncles, the pons, and the medulla oblongata, and since his time our knowledge of the process has been greatly advanced by the investigations of Turck,[2] Waller,[3] Van der Kolk,[4] Phillipeaux and Vulpian,[5] Gubler,[6] and Bouchard.[7] Laborde[8] has, moreover, quite recently stated that in cases where there is softening of the corpus striatum or of the optic thalamus, a similar process is also set up on the surface of the hemispheres in some related portion of the superficial grey matter of the convolutions. These softenings of the convolutional grey matter are stated to be always on the same side of the brain as the lesions in the central ganglia, and Laborde says he has also ascertained that a relationship exists between the particular convolutions affected and the particular portions of the central ganglia which have been destroyed, so that where softening of the anterior portion of the corpus striatum or optic thalamus exists, the same process occurs on some portion of the anterior convolutions; with destructions of the central portions, the middle convolutions are affected; and with destructions of the posterior portions of either of the central ganglia, a corresponding change is set up in some of the posterior convolutions. Should future observations confirm the opinions of Laborde, these changes would seem to be related to the secondary atrophic degenerations, and would be most interesting in a physiological as well as a pathological point of view: it is well to mention, however, that MM. Vulpian and Charcot maintain[9] that the coexistence of these peripheral and central lesions is a mere coincidence, and that there is no necessary association between them.

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[1] Anat. Patholog.

[2] Comp. rend. Acad. des Sciences de Vienne, Mars, 1857.

[3] Nouv. Méth. Anat. pour l'Investig. du Syst. nerv. (Lett. à l'Acad. des Sc. 1852.)

[4] New Syd. Soc. vol. xi. p. 129.

[5] Mém. de la Soc. de Biolog. 1859, p. 343.

[6] Archiv. gén. de Méd. 1859, p. 31.

[7] Ibid. Mars, 1866.

[8] Le Ramolliss. et la Congest. du Cerveau, Paris, 1866.

[9] Physiolog. génér. et comp. du Syst. nerveux, Paris, 1866, p. 653.

Seat of Cerebral Softening.—Foci of softening may be found in all parts of the brain, but they exist most frequently in the convolutions and most rarely in the cerebellum and in the pons Varolii. Durand-Fardel[1] found, as the result of an analysis of his own observations combined with those of Rostan, Andral, Raikem, and Lallemand—yielding altogether eighty-six cases of "acute" softening—that the convolutions were affected fifty-nine times; the corpus striatum and optic thalamus, either singly or combined, twenty-eight times; and the white substance of the hemispheres, alone, only nine times. Although the combined statistics yield this result, however, it does not agree with the experience of Rostan[2] alone, who says expressly that the corpora striata and the optic thalami are the parts most frequently affected with softening, and that after them comes the central white substance of the hemispheres. Laborde says[3] that the corpus striatum is affected nearly twice as frequently as the optic thalamus, since he has found the former softened forty-six times and the latter only twenty-four times. He states also, that the change most frequently exists only on one side, and that it is rare for the two corpora striata to be affected at the same time without a corresponding change in one or other of the optic thalami. The two hemispheres are about equally liable to undergo such a process, though, as before indicated, the softenings of embolic origin seem to occur rather more frequently in the left hemisphere.

The extent of the softened patch is extremely variable: it may be found from the size of a pea up to such an extent that the whole of one lobe, or the greater part of one hemisphere even, may be so affected.

Periods of Life at which Softening of the Brain is most frequent.—Durand-Fardel has attempted to show by a combination of his own cases with those of Rostan, Lallemand, Bouillaud, and Andral, that Softening of the Brain may be met with at all periods of life, and that it is by no means a malady peculiar to old age. Laborde,[4] however, has subjected the same statistics to a more rigid scrutiny, and by eliminating various cases of inflammatory softening of traumatic origin, and others which have been improperly included from various causes, he has found that the very statistics made use of by Durand-Fardel do show, if properly sifted, that the most undoubted relationships exist between non-inflammatory Softening of the Brain and old age. In a total of eighty-one cases, seventy-five of the individuals were from sixty to seventy-five years of age, four from fifty to sixty, whilst only two individuals were between thirty and fifty years of age. It must not, however, be supposed that Softening of the Brain is so exclusively a disease of old age as these figures would represent, since softenings of embolic origin are now known frequently to occur in young adults and even in children; softenings from thrombosis, also, may and do occur occasionally in certain adults (at any age), who may be suffering from cachectic diseases. Still, degeneration of the cerebral arteries and the various other causes of thrombosis increase in frequency with increasing age; and so is it with non-inflammatory softenings of the brain, to which these conditions are bound in such a close pathological relationship.

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[1] Malad. des Vieillards, Paris, 1854, p. 68.

[2] Ramolliss. du Cerveau, Paris, 1823, p. 161.

[3] Loc. cit. p. 72.

[4] Loc. cit. pp. 144–185.

MORBID ANATOMY.—Softenings of the brain present themselves under different forms according to their variations in seat, their age or duration, and other conditions; and the common character of diminished consistence, which first attracted the attention of pathologists, is curiously enough the one least indicative of a morbid change that has taken place during life, since marked alterations of this kind may be induced *post mortem.* The tests employed by earlier pathologists, therefore, which were intended to establish this one fact of diminished consistence, namely, the effect of a gentle stream of water in washing away the softened tissue, the impressions derived from the sense of touch, and the speedy rounding of the angles which takes place in a portion of brain cut out from the softened portion, are useless for the purpose of demonstrating that we have to do with a pathological process established during life. To make sure of this we must take into account other characters afforded by the softened tissue, and have recourse to more delicate means of investigation, such as are afforded by the microscope and the specific gravity apparatus.

The most notable differences of appearance in softenings of the brain are due to variations in colour. The greatest variety of tints may be met with. In some cases the softened tissue has a dead white colour; or it may be of a reddish hue, and may present all shades from a uniform pinkish tint, through different grades of red, up to a claret colour; whilst, in other instances, various shades of yellow and even brown discoloration are met with. These colours may exist alone, or variously intermixed. But in spite of this apparent complexity, there are three principal kinds of "ramollissement"—known as the *white*, the *red*, and the *yellow*—under which heads it will be convenient for us to describe the various forms of Softening of the Brain. But in making this division it should be understood that we do not necessarily look upon these varieties as different stages of the same condition, as has been done by Durand-Fardel[1] and Lancereaux[2]—both of whom describe "ramollissement blanc" as the last or chronic condition of red softening. Lallemand also looked upon white softening as due to a purulent infiltration, though the absence of pus has been established again and again by later observers. On the other hand, MM. Hardy and Béhier,[3] as well as Rostan and Abercrombie, have pointed out that this kind of softening is often met with in old people, and more particularly in those who are weakened and feeble, which agrees well also with the observations of M. Charcot, who has often met with white softening in old people afflicted with cancer at the Salpétrière.[1] MM. Prevost and Cotard, moreover, admit the existence of a Softening of the Brain, white from the first; and this has also seemed to be the case in several instances which have fallen under our own notice. It would appear probable that white softening is mostly due to a rather more chronic process than that which gives rise to the red, and that it is such as would exist if this change had been brought about by disease of the coats of the capillaries, supplemented by general mal-nutrition from poverty of blood. Prevost and Cotard [2] suggest, also, that in some of those cases, where the softening seems to be associated with obliteration of vessels, the absence of redness may be due to the extent and nature of the arterial obliteration being such as to prevent the collateral fluxion of blood into the vessels of the diseased part. In other cases they admit that the cause seems inexplicable. Red softening seems to be more particularly limited to cases in which there is arterial obstruction, or impediment on the venous side of the circulation; whilst there can be little doubt that many of the shades of yellow in softenings of the brain are due to alterations that have taken place in what was previously red softening, although Rokitansky has described a special kind of yellow softening having intrinsic characters of its own.

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[1] Malad. des Vieillards, Paris, 1854, p. 72.

[2] Loc. cit. p. 20.

[3] Traité de Pathol. int., Paris.

1. *White Softening.*—In this species of ramollissement, which occurs only in the white substance of the hemispheres, a varying amount of diminution of consistence is met with, without alteration of colour. In some cases the amount of softening is so slight as to escape detection by the unaided senses, whilst in others the portion of brain is reduced to a thin diffluent pulp, which may be poured out so as to leave a distinct cavity with irregular though softened walls—for the degeneration is never strictly limited, it shades off imperceptibly into the healthy tissue. Thus, in what appear to be minute circumscribed patches of white softening, granular corpuscles are diffused for some distance in the firmer tissue surrounding the softened patch. Sometimes nearly the whole of the white substance of one hemisphere may be found in a more or less softened condition. The specific gravity of softened white matter usually falls to 1·032, but when in its natural condition it is about 1·040; and as a general rule it may be said that any portion of the brain when softened has a specific gravity lower by six to eight degrees of the hydrometer scale than the specific gravity of the same part when in its normal condition.[3]

2. *Red Softening.*—This variety may be met with either in the grey matter or in the white substance of the brain, though in the former the coloration is generally more distinct and of a darker hue, on account of the naturally greater vascularity of the brain substance

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[1] Vide Proust, loc. cit. p. 49.

[2] Gaz. Méd. de Paris, 1866, p. 207.

[3] "On the Specific Gravity of different parts of the Human Brain:" Journ. of Ment. Sc., Jan. 1866.

in this situation. When affecting the grey matter, the patches may be small and distinctly circumscribed, so as to present the appearance of superficial ulcerations; or they may be more diffuse and extensive, presenting less of the appearance of ulcerations, but existing as dark, somewhat swollen patches, which from their colour were formerly spoken of as "plaques ecchymotiques," and "taches scorbutiques des convolutions." These have also been described by Cruveilhier[1] under the name of "ramollissement *hortensia* ou *lilas*." This softening of the convolutional grey matter may exist alone, it may be associated with a similar change in the subjacent white matter, or, as Laborde has pointed out, it may coexist with softening of one of the central ganglia on the same side. In these latter cases the softening is often slight, and takes place without much alteration in the natural colour of the part, so that it is liable to be overlooked. Where the convolutions are softened, the grey matter is frequently torn in attempting to strip off the membranes. This may be due, however, to the mere fact of diminished consistence, rather than to any increase in the natural adhesions existing between the parts. Red softening of the white matter often exhibits a general rosy hue, intermixed with darker-coloured patches from effused blood and minute points of redness, which have been described as "capillary apoplexy." It may be diversified also with patches of simple white softening here and there, and after a certain time shades of buff, yellow, and even brown coloration supplant or become mixed up with the red. The brown colour is most frequently met with in old patches of softening in the corpus striatum. The red colour being due to the increased quantity of blood in the part, owing to extreme congestion of the vessels (combined with staining from transudation of colouring matter), we have, in this fact, an explanation of the circumstance that when a recent red softening is cut into, the surface often rises up above the level of surrounding parts, and presents a slightly swollen appearance. Also, when red softening of the white matter exists combined with a similar condition of several of the contiguous convolutions, owing to the swelling and consequent pressure thus produced, these become flattened on the surface, whilst the sulci are rendered indistinct. The process of degeneration of the nerve tissue is the same in this form as in the simple white softening, and it may advance to the same condition of diffluence. It is only that the colour and composition of the softened part in a case of red softening are altered by the great admixture of blood together with the products of its retrograde metamorphosis.

Red softening, if not of traumatic origin, as before stated, is almost invariably connected with obliteration of the vessels or other impediment to the circulation, and the "redness" and "swelling," which were formerly considered so indicative of its inflammatory origin, are capable of receiving an altogether different explanation, tending to show that these characteristics are dependent upon mechanical rather than upon vital influences.

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[1] Anat. Patholog. vol. i. Livraison.

It has been known for some time through the writings of Rokitansky and Cohn,[1] that infarctus of the abdominal viscera, owing to arterial obstruction, commences as a deep red spot in the territory of the affected artery, this appearance being due to the engorged condition of its capillaries, and the occurrence of minute extravasations of blood. And, quite recently, MM. Prevost and Cotard having injected into the arterial system of a dog (whose abdominal walls had been opened so as to expose its contained viscera) some water holding tobacco-seeds in suspension, in a few moments after the injection there appeared, at the inferior extremity of the spleen, a red prominent spot of definite outline, which rapidly increased in size till it equalled that of a two-franc piece. The corresponding branch of the splenic artery was afterwards found to be obliterated by some of the seeds. Ecchymotic-looking spots were produced upon the kidneys at the same time. What was here actually seen to take place accords perfectly with what is observed in cerebral softenings brought about by the injection of foreign bodies into the arterial system. Their impaction in the cerebral arteries constantly results in the production of red softenings with great fulness of the vessels, as has been abundantly proved by the experiments of Cohn, Vulpian, and by those of MM. Prevost and Cotard. Here, then, we have the two appearances, redness and swelling, produced so rapidly as to make it quite out of the question for us to regard them, in spite of the opinion of Oppolzer, as of inflammatory origin; so that we must seek for some mechanical cause of the phenomena. Cohn and Rokitansky attribute the results to a collateral fluxion of blood through the contiguous capillaries, whilst Virchow regards them as the effects of a venous reflux, in consequence of the suppression of the *vis à tergo* on the side of the artery. In support of the former view, Weber[2] has since pointed out that when an artery is obliterated, the pressure at the point of obliteration increases, and becomes equal to that at the origin of the artery, and that when a certain number of capillaries are obliterated, the pressure augments in the artery belonging to them, and in those remaining pervious. Prevost and Cotard also support this view, and they call attention to the theorem of hydrodynamics upon which it depends, to the effect, that when a tube receives at one of its extremities a fluid at a certain pressure, and allows it to escape freely at the other extremity, the pressure diminishes from one end to the other of the tube according to an arithmetical progression. Marey[3] has shown that this theorem is applicable to the circulation of blood, owing to the resistance in the veins being so slight as practically to make it appear as though there were a free flow into the capillaries.[4] This increased tension of blood in the collateral capillaries would, therefore, seem to account in great part, not only for the surrounding congestion, but for the flux of blood into the territory of the obliterated artery, where it would stagnate; and, owing to the increased tension of the blood (combined with the progressive weakening of the capillary walls, and the diminution of their usual support from the softening of the surrounding brain tissue), we can easily conceive the mode of origin of those effusions of blood and those dilatations of capillaries such as are actually encountered.

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[1] Klinik der Embolisch. Gefässkrankh., Berlin, 1860.

[2] Handbuch der Allgem. und Spec. Chirur. 1865.

[3] Physiolog. Méd. de la Circ., Paris, 1863.

[4] This is an important point, because Poiseuille (Recher. sur les Causes du Mouv. du Sang) maintained that the pressure was the same in all parts of the arterial system.

3. *Yellow Softening.*—A special form of degeneration of brain substance has been described under this head by Rokitansky.[1] It usually occurs in sharply circumscribed spots—varying in size, but rarely exceeding that of a hen's egg—in which the cerebral substance is converted into a very moist, tremulous, and occasionally gelatinous pulp, retaining none of the characters of proper brain tissue. The altered portion rises considerably above the level of the section, and is of a straw or sulphur-yellow colour. In slighter degrees of the disease, the colour is merely dull white, inclining to yellow, though the tissue is still much softer and moister than usual. The expressed fluid has a distinctly acid reaction. The transition to the healthy brain tissue is usually abrupt, and there are no signs of inflammation in or around the softened patch. Its usual seat is the white matter of the hemispheres, though it may affect the central ganglia, or, much more rarely, the convolutional grey matter. With regard to the pathogeny of this form of softening we are almost entirely in ignorance. Rokitansky looks upon it as a peculiar chemico-pathological transformation of brain substance, in which "the liberation of an acid—the phosphoric, and especially one or more of the fatty acids—may be conjectured to be one of the most important phenomena." Besides occurring as an independent condition such as above described, this form of yellow cerebral softening is said by Rokitansky to exist frequently around old clots, tumours, or other adventitious products in the brain. Cruveilhier[2] has also frequently met with it around old adventitious products, though he has never seen it existing alone.

Besides this special form of yellow softening, as before stated, we meet with a yellow colour of the brain substance in the secondary stage of red softening, in which case the tint may be partly due to the presence of altered blood pigment, and partly, according to Lancereaux and Virchow, to the presence of what were the red globules of the blood, but which have now, in part, been deprived of their hæmatine.

Histological Alterations and Microscopical Appearances in different Stages.—The changes which take place in the nerve elements of a part, whether from the separation of these from their proper ganglionic connexions, or from the cutting off of the supply of blood to the part, have been shown to be absolutely identical in nature, and to differ only as regards the rapidity with which the change is brought about. This has been proved by the experiments and observations of Phillipeaux and Vulpian on the changes taking place in the peripheral

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[1] Path. Anat. (Syd. Soc.) vol. iii. p. 419.

[2] Anat. Path. vol. i. Livraison viii.

extremities of divided nerves, and by the observations of Turck, Gubler, and Bouchard on the progress of secondary degenerations of the spinal cord, as compared with the ordinary histological changes which take place in softening nerve substance. In the one case we have a simple destruction, or necrobiosis, taking place amongst the elements of the tissue, in which, from the comparatively slow way that they are brought about, all the steps of the process may be easily traced, whilst in the other we have a more rapid and tumultuous form of necrobiosis, which, on account of its rapidity, is associated with diminished consistence. The investigations of MM. Phillipeaux and Vulpian[1] have yielded the following results. At the end of the first day there is found to be a diminution of the proper excitability in the peripheral extremity of the cut nerve, whilst this is lost altogether at the end of the fourth, and the filaments which had previously shown no change begin to alter in appearance. From the fifth day the medullary substance of the tubes seems to be coagulated, and at the same time fissures establish themselves in its thickness and divide it into unequal blocks or divisions. This is called the segmentation of the nerve tubes. Soon after this the fragments of the medullary substance undergo a further alteration, fatty granules[2] form in their interior, which go on increasing in quantity, and at last entirely replace the fragments of myeline. Bouchard[3] has recognised precisely the same changes in cases of secondary atrophic degeneration, and, speaking of one of these cases, he says:—"Independently of the fatty granules contained in the altered tubes, a great number were free between the tissue elements, and, at certain points, aggregated together into masses, so as to constitute what are known as the 'corps granuleux' of Gluge." In the same case, the vessels presented on their surface heaps of molecular fat particles, or even a complete envelope of these, so as to render the vessels black and opaque under the microscope. Here, then, are produced, without the intervention of inflammation, all the appearances which have been supposed to be characteristic of inflammatory Softening of the Brain. Dr. Hughes Bennett says:[4]—"*Exudative or inflammatory softening* always contains granules and granule cells, which are numerous, according to the degree of softening. The granules are for the most part seen coating the vessels, and the cells also may occasionally be seen there in various stages of development. In the demonstrations that are made under the microscope, they are frequently seen diffused among the tubes, which, according to the severity and extent of the lesion, are easily separated from one another, or broken up in a variety of ways."

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[1] Mém. de la Soc. de Biologie, 1859, p. 343.

[2] At the end of a certain time the fatty granules are absorbed, and nothing is left but the sheath of Schwann folded on itself and on the axis cylinder. Little is known as to the actual condition of this last, though the researches of Schiff and MM. Phillipeaux and Vulpian go to prove that the return of function in a divided nerve proceeds not from the production of new tubes amongst the débris of the old, as formerly supposed, but from the reformation of myeline within the wasted sheaths themselves, and around the old axis cylinder, which is capable of persisting for a long time without undergoing much appreciable alteration.

[3] Arch. Gén. de Méd. 1866, p. 281.

[4] Clinical Lectures. Fourth Ed. 1865, p. 354.

MM. Prevost and Cotard have found from their experiments on dogs, that at the end of the first twenty-four hours after the obstruction of an artery there was red pulpy softening, with slight diminution of consistence, and, on examination with the microscope, there were seen broken-up fragments of nerve tubes, drops of myeline, blood corpuscles, and peculiarities of the capillaries, though no granules or granular corpuscles were at that time visible. As early as the third day, however, they have found granule corpuscles formed, and an abundance of granular matter lying amongst the tissue elements, as well as more especially aggregated along the walls of the vessels. These results are quite in accordance with our own observations, since we lately met with an instance of traumatic softening in which a few fully developed granule corpuscles, and very many in a less mature state, were seen, which must have been produced in rather less than two and a half days.[1] Bouchard believes that these granule corpuscles may result from the "granulo-graisseuse" degeneration of drops of myeline, and Prevost and Cotard also think they may result from the aggregation of granules originally separate. We have, ourselves, never been able to substantiate either of these modes of origin, and we agree with Virchow[2] in the opinion that they mostly originate from the fatty degeneration of the cells of the neuroglia, since granule corpuscles are commonly met with in the midst of the white matter of the hemispheres, having a more or less distinct cell wall, and which show a large nucleus in their interior after staining with carmine.[3] The cells of the neuroglia are the only elements existing in this situation capable of giving rise to such bodies.[4] They are also to be seen in the grey matter lying between the ganglion cells, which, in old age, undergo more or less of the pigmentary degeneration, and always present quite a different appearance. Robin formerly held that these granular corpuscles were produced by the degeneration of pus cells; but pus cells in their natural state are never met with in simple Softening of the Brain, and it seems scarcely fair or reasonable to assume that they should be seen only in a state of degeneration. In cases of softening of the convolutional grey matter or of the central ganglia, a degeneration of the proper nerve cells takes place, which become filled with dark-coloured granules. These are generally at once distinguishable from ordinary granule corpuscles by their irregular, angular shape, and by the presence of the stumps of one or more cell prolongations. At the same time that these bodies are forming in the degenerating tissue, granules collect along the walls of the capillaries, partly in an altogether irregular manner, and partly in the form of more or less spherical aggregations. Some of the capillaries become completely covered in this way; but the collection of granules is *on* the walls of the capillaries, and is a consequence, not a cause of the softening. It must not be confounded with fatty degeneration of these vessels, in which the granules are imbedded *in* the walls of the capillaries. There are other alterations of the capillaries met with, especially in red softenings, which have been particularly dwelt upon by Laborde.[1] At first, partial dilatations of the walls of the capillaries are seen, like minute aneurysmal swellings, or, in other places, little ampulliform dilatations including the whole circumference of the vessel, and constituting what he describes as the *moniliform* condition of the capillaries. At a later stage, complete as well as partial dilatations of the capillaries are to be seen, together with actual ruptures here and there, and minute extravasations of blood. Still later, the capillaries become enormously dilated, and their walls thin and granular from degeneration. The punctiform hæmorrhage, to which Cruveilhier gave the name of "apoplexie capillaire," is sometimes due to minute extravasations from rupture of the capillaries, sometimes to the extreme dilatation of capillaries gorged with blood, and often to the production of what has been wrongly called "dissecting aneurysm," occasioned by rupture of the proper wall of a minute artery and an effusion of blood into the lymphatic sheath which surrounds it. The blood remaining in the capillaries, and also that effused externally amongst the nerve elements, shows, for a certain time, traces of the individual blood corpuscles, more or less decolorized and yellow, as well as flattened and pressed together; whilst mixed up with them are reddish or reddish yellow flakes of tissue, stained by the transuded hæmatine. In those minute patches of extravasated blood, in which the colouring matter exists in some quantity, we afterwards find it in the form of amorphous, yellow or orange-coloured granules or flakes, intermixed with the characteristic orange or ruby-coloured crystals of hæmatoidine. These are very minute, and of an oblique rhomboido-prismatic form. It is not known exactly in how short a time these crystals may appear in extravasations of blood in the human brain. Dr. Wilks[2] has, however, met with them as soon as three weeks after such an occurrence, and Cruveilhier found the "coloration jaune orangé" developed after twenty-five days in the seat of an hæmorrhagic effusion into the brain. Once formed, the hæmatoidine crystals remain as indelible evidences of past extravasation of blood.

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[1] The man on whom this observation was made fell down an area and fractured his skull. He was admitted into St. Mary's Hospital on Sept. 7, 1866, at 4 P.M., immediately after the accident, and died on the 10th of the same month at 3.10 A.M. The exact interval was, therefore, 2 days, 11 hours, 10 min.

[2] Wiener Medicin. Wochenschr. January 19, 1861.

[3] Case of Concussion Lesion, Med.-Chir. Trans. 1867.

[4] Corpuscles almost precisely similar are met with in other organs, whose tissues are in a state of degeneration, which undoubtedly originate from the fatty and granular degeneration of pre-existing cells.

In the extreme stage of softening, the fluid matter occupying its site no longer presents the slightest trace of nerve structure—the degeneration is complete, and nothing can be recognised by the microscope save granules and granule cells, mixed up with the various kinds of blood pigments, amorphous fragments of tissue, and the débris of degenerated vessels. When we have to do with the last stage of red softening, and especially when this is situated in the corpus striatum or optic thalamus, the contents of the softened centre may present a brownish or even chocolate hue.

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[1] Loc. cit. p. 114.

[2] Lect. on Path. Anat. 1859, p. 133.

Fatty degeneration of the tissues being complete, the process of repair begins at a variable period—probably in from one to two months after the commencement of the degeneration. These alterations have been fully described by Durand-Fardel,[1] and differ according as they are situated at the surface of the brain, or in its central parts. In the former situation the process results in the formation of the so-called "plaques jaunes," and in the latter it is accomplished by what Durand-Fardel calls "infiltration celluleuse."

These so-called "plaques jaunes," which have been well represented by Cruveilhier,[2] exist in the form of yellow or ochre-coloured, rounded patches. They may be confined to a single convolution, or may extend over several, at the same time dipping down into the sulci. The pia mater over them may sometimes be easily stripped off, whilst at other times it is closely adherent to the tissues beneath. The substance of the patch, though pliable, is tough and resists the knife; it usually implicates the cortical grey matter only, and its circumference is pretty sharply defined from the surrounding healthy tissue. More rarely, however, it is separated, as well circumferentially as beneath, from the healthy brain substance, by a layer of softened tissue. Histologically, these patches are composed of connective tissue containing an abundance of nuclei; also of intermingled hæmatine granules and crystals of hæmatodine, together with fatty particles, a few granule corpuscles, and some degenerated vessels. Rokitansky[3] denies that these yellow patches are the sequelæ of softening of the convolutions, and looks upon them as changes resulting from superficial hæmorrhage. But the result of a recent experiment by Prevost and Cotard goes strongly to support the view of Durand-Fardel. They found a well-marked yellow patch, on the middle lobe of one of the hemispheres of a dog, which was in every way similar to those met with in man; and the corresponding middle cerebral artery of this dog had been obliterated, thirty-five days before the death of the animal, by the injection of tobacco seeds into the carotid artery. From what we ourselves have seen, however, we are inclined to think that superficial extravasations of blood into the pia mater may also, as Rokitansky says, give rise to yellow patches, though of a different kind from the "plaques jaunes" described by Durand-Fardel. In cases where a superficial hæmorrhage has been the antecedent condition, the coloration is almost entirely due to an accumulation of blood pigment in the meshes of the pia mater, with atrophy of the subjacent convolution, rather than to a fibro-cellular conversion of the substance of the grey matter itself.

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[1] Malad. des Vieillards, Paris, 1854, p. 72.

[2] Anat. Path., Livraison 33, pl. 2.

[3] Patholog. Anat. (Syd. Soc.), pp. 394 and 416.

When a focus of softening in the midst of the white substance of one of the hemispheres begins to undergo the process of repair, the walls of the softened cavity become bounded by a pulpy tissue of a white or greyish colour, which, on microscopical examination, is found to be continuous with the neuroglia of the contiguous healthy portion of the hemispheres. Tissue of the same kind also extends across the cavity in different directions, breaking it up into divisions or compartments, in the meshes of which may be found a whitish liquid containing fragments of nerve substance which have not yet completely undergone the fatty metamorphosis. This fluid holds in suspension, also, fat particles, and a number of corpora amylacea. The formation and growth of the vascular and nucleated connective tissue constitutes the "infiltration celluleuse" of Durand-Fardel. Though met with principally in the white substance of the hemispheres, it is also seen more rarely in the central ganglia. When situated in the corpora striata, the walls, instead of being white, are often of a yellowish or ochre colour, which makes the identity of this process with that which gives rise to the "plaques jaunes" of the convolutions all the more evident. The fluid contents of the cavity gradually become absorbed, and its walls close in and contract in the same way as do those of an apoplectic cyst. Indeed, in these last stages there may be some difficulty in discriminating between the two. In the remains of the apoplectic cyst, however, more colouring matter is usually found; its walls are also generally more dense and contractile, and a more complete obliteration of the old cavity is said to follow.

Lastly, there is a condition of the central ganglia of the brain, and more particularly of the corpora striata, which has been described by Durand-Fardel[1] under the name of "état criblé." On making a section of these central ganglia, small pisiform cavities or lacunæ are occasionally seen, which sometimes seem bounded by a distinct membrane. Similar cavities may also be seen, though more rarely, in the pons Varolii. These are regarded by Laborde[2] and others as minute apoplectic cysts, resulting from slight effusions of blood, whilst others again look upon them only as dilatations of the lymphatic canals, in which, as pointed out by His,[3] the cerebral vessels are contained. Laborde, however, thinks there is another and more important modification of this condition, in which no lining membrane is to be met with, but in which the little cavities are somewhat larger, so as to be even capable of containing a good-sized pea. These he looks upon as the result of "une désorganisation partielle et progressive," and as true, though minute and circumscribed, softenings of the parts in which they are found.[4]

[1] Who, however, attaches little importance to this condition, and looks upon the little cavities as the results of dilatations of the vessels, owing to long-continued congestion.

[2] Loc. cit. p. 94.

[3] Zeitsch. für Wissen. Zoolog. 1865. Bd. xii.

[4] Laborde says: "Nous possédons plusieurs observations de ces curieuses désorganiations partielles siégeant au centre de la *protubérance annulaire* et paraissant répondre

It only remains for us now to notice the softenings which have a post-mortem origin, and to point out how these may be distinguished from those having a real pathological significance which we have hitherto been considering.

Ordinary post-mortem softening of the brain is due to the combined influence of two causes; namely, putrefactive changes, and the maceration of the cerebral tissue from absorption of fluid.[1] This is commonly met with on the surface of the thalami and in the parts bounding the posterior portions of the lateral ventricles, in all those cases where an interval has existed between the death and the autopsy, and more particularly when the atmospheric temperature has been high and the ventricles have contained an excess of fluid. In these cases the surface of the parts affected is broken up, and presents an irregular appearance, whilst the tissue itself is in a more or less diffluent condition. The fornix also frequently shares in this change. It has been a subject of dispute as to what is the nature of the process which gives rise to the softening of the central parts of the brain in acute hydrocephalus—whether, in fact, it has been produced by inflammation, or is merely the result of maceration; and in the event of the latter method of pathogenesis being the real one, whether this maceration has occurred during life or after death. Doubts have been expressed by many pathologists as to whether such a process of maceration ever occurs during life.[2] When merely macerated nerve tissue is examined by the microscope, broken up and dissociated nerve elements only are met with, and none of the granule corpuscles or other appearances characteristic of real softenings that have been produced during life. Examined by the specific gravity apparatus also, we have several times found the actual density of the altered tissue the same as that of contiguous unaltered portions. This is somewhat remarkable and becomes very characteristic; since if a portion of brain tissue having a similarly diminished consistence, brought about by a pathological softening rather than by a post-mortem maceration, had been examined, the specific gravity would have been found lower than that of similar healthy tissue in the same brain by from eight to ten degrees of the hydrometer scale. The specific

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au point de vue symptomatique, à certains cas de paralysie diffuse, généralisée, dans laquelle s'éteignent progressivement un grand nombre de vieillards."— P. 95.

[1] Dr. Bennett calls attention, in his "Clinical Lectures," to the softenings which may be produced by mechanical means, owing to the clumsy use of instruments in removing the brain and spinal cord from the body. This mode of origin should also be borne in mind.

[2] The impediment to the return of blood through the venæ magnæ Galeni, owing to thrombosis in these vessels, to which the collection of fluid in the ventricles is in part due, also gives rise to a condition of œdema in the walls of the ventricles themselves, and is followed by a true degenerative softening of the brain tissue. (Pathology of Tuberc. Mening., Edinb. Med. Journ., April 1867.) Respecting this condition of œdema of the brain, however, which it may be presumed occurs occasionally in heart disease and other conditions impeding the return of blood from the head, we have no very definite knowledge. It seems doubtful whether any amount of serous infiltration would be capable of producing actual softening during life, or do more than make the brain appear flabby—a little moister and softer than usual—and, at the same time, slightly lower its specific gravity. The brain is usually said to be "wet" when in this condition.

gravity test thus becomes a most important auxiliary to the microscope; and we have several times found it most useful in examinations of the spinal cord. Thus, a short time since, on making sections of a cord through the cervical, dorsal, and lumbar regions respectively, the surfaces exposed were quite pulpy and irregular in the two former regions, whilst in the lumbar portion the surfaces were firm and smooth. Yet the specific gravity of portions of the cord from the dorsal region was the same as that of other portions from the lumbar region, whilst in the cervical region the specific gravity was even slightly higher. Microscopical examination, moreover, yielded no evidence of a pathological change in any portion of the cord. We have found much the same state of things also in other cases. Diminished consistence or diffluence, therefore, must not be confounded with diminished density or specific gravity; and it should be remembered that it is the combination of the two, associated with certain microscopical changes, which are the characteristics of real pathological softening of the brain.

DIAGNOSIS.—1. *Acute softening* may, in its *apoplectic form*, be confounded with congestion of the brain, with hæmorrhage, or with urinæmia; but by regard to the mode of onset of the symptoms, and to their proportion *inter se*, a diagnosis may be established in the majority of cases. At the onset of attack it may be impossible to distinguish the nature of the malady, but after a few minutes, or perhaps only after two or three hours, it is possible to aim at something like certainty.

From *congestive apoplexy* softening may be distinguished by the longer duration of mental obtuseness; and by the distinct limitation of intellect in one or two directions, when the general obscuration of the "fit," or "stroke," has passed away. At the onset of attack in congestive apoplexy there may be complete loss of consciousness, and the same thing may occur at the commencement of acute ramollissement, and for precisely the same reason, viz. the presence of congestion. In the former case, however, the mind rapidly recovers, and is restored to its previous condition; in the latter all that was due to mere congestion is speedily removed, but there remains the impairment due to softened tissue. This may be aphasia, or some other special alteration in the mental powers, such as have been described in the section upon symptoms. Attacks of softening differ still more distinctly from those of congestive apoplexy, when they are unattended by any of the phenomena of hyperæmia, and occur after middle life, and especially in advancing years. The patient becomes more or less suddenly confused, but does not lose his consciousness; he may wander in his talk, utter some exclamation of alarm, or may simply look distressed and as if about to cry; he knows what is said, and makes signs to those about him; is obviously aware that something very wrong has happened, and continues in this state of mental impairment for hours, weeks, or months. The difference from congestion is seen in the primary absence of general mental change,—loss of consciousness,—and in the persistence of limited intellectual failure.

The two classes of change in function, now described with regard to mind, are to be observed also in respect of sensation and motility. There may be, at the moment of attack, general anæsthesia and general paralysis; but if so, they are due to congestion or to shock, and they, with either of those conditions, soon pass away; leaving behind them, however, impaired sensation in one or two limbs, and with this, localised paralysis. On the other hand, there may be no general change in the power of feeling or of motion—there may be neither congestion, nor sufficient shock—the patient feels some numbness, coldness, or deadness, together with weakness of one or two limbs, and these conditions pass on into hemiplegia, *i.e.* loss of power, and loss or diminution of sensibility in the arm and leg of the side opposite to the lesion. Beyond these facts there is not unfrequently some hyperæsthesia or morbid sensibility of the paralysed limb, and this is much more common in cases of softening than in those of congestion; and, again, there is twitching of the limbs, or rigidity of those that are paralysed.

The general aspect of the patient differs from that of the person struck with congestive apoplexy. There is, unless congestion be present as a transient condition, pallor instead of dusky redness, coldness of the head instead of heat, and a faint look in the place of bloated suffocation; there is often some sweat upon the brow; the patient is spare in habit, and the attack occurs when sitting quietly at the desk, or when making no such exertion as could tax the physical powers. Beyond these general conditions there may be observed rigidity of arteries, an irregular, weak, and often intermitting pulse, a feeble heart, arcus senilis, and irregular distribution of bodily warmth.

From *hæmorrhagic apoplexy* acute softening may be sometimes distinguished by the following considerations:—In hæmorrhage there is often some evidence of either congestion or of shock; in softening there may be an entire absence of both. In hæmorrhage the attack frequently occurs at night; the patient goes to bed apparently well, and wakes in the morning feeling as usual, but on attempting to move finds that one side is paralysed. In hæmorrhagic apoplexy the attack is often absolutely instantaneous, in softening it is gradual. In the former there may be not the least—even momentary—confusion of mind, whereas in the latter there is distinct mental perturbation and insufficiency. In hæmorrhage, when the intellect is profoundly affected, as it sometimes is at the onset of attack, there is often a rapid restoration, and in the course of a quarter of an hour the patient's mind is as clear as it was before; in softening *per se* there is less distinct mental obscuration at the commencement, and little or no subsequent recovery. In hæmorrhage there is sometimes alarm, and not unfrequently anxiety and depression, whereas in softening there is more commonly too much confusion of thought for any definite apprehension to be entertained, and sometimes there is transient excitement or mild delirium.

Sensibility is often unaffected in hæmorrhage, it rarely escapes altogether in an attack of softening. In the former there is, as a rule, unilateral anæsthesia, which rapidly diminishes or disappears; in the latter there is dulness of sensation, with morbid feelings of coldness, numbness, tingling, &c. which persist.

The paralysis in hæmorrhage is, typically, hemiplegic; in softening it is more irregular, and sometimes more closely limited. In the former there is neither rigidity nor convulsion unless the coma be profound, and the paralysis extensive; in the latter there is often either twitching or tonic spasm even when the paralysis is slight, and the mental perturbation comparatively trivial.

In hæmorrhage there is very frequently hypertrophy of heart, with granular degeneration of the kidneys; in softening there is very commonly a weakened heart, with valvular disease.[1] Hæmorrhage may occur in a person of strong limb and general good health, whereas softening is more common in the aged, the enfeebled, and those who have suffered from exhausting diseases, or still more exhausting cares.

From *urinæmia* as a cause, *per se*, of an apoplectic seizure, softening may be distinguished by the fact that in the former, convulsions of some kind and to some degree almost invariably precede the coma; and that these have followed premonitory symptoms of drowsiness, oppression, and headache. The coma exhibits in a marked manner the features sometimes observed in acute softening of the brain, viz. an apparent profundity, with susceptibility of being roused. Urinæmic patients lie in apparently profound torpor, but they may be roused by a touch or a word, and may appear in possession of all their faculties; left to themselves they relapse almost instantly into the state of stupor. In softening, if the coma be highly marked the awakening is less distinct, and the answers are less rational. In urinæmia there is often amaurosis, and a generally obtuse condition of sensibility, neither of which are frequent in ramollissement. There is much twitching of the limbs, and often marked rigidity in the former case, but the locality of these symptoms changes from side to side, and is not accompanied by fixed paralysis; there may be twitching or rigidity in the case of softening, but these are found in the same limbs day after day, and are attended by distinct and persistent loss of power. The stertor in urinæmia is unlike that of softening, being oral rather than guttural;[2] the pulse-respiration ratio is much changed, being sometimes 5 : 1;[3] sensori-motility and irritability of muscles on percussion are often notably increased. Beyond these features there are the signs of characteristic debility and cachexia, the pale waxy skin, with vomiting and diarrhœa; and above all the œdematous condition of the eyelids and ankles, together with albuminous urine. In cases of acute cerebral softening, all these symptoms may be absent. The two diseases are frequently combined, but when they are so it is not impossible to determine how much is due to the one, and how much to the other condition.

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[1] Dr. Kirkes found the heart hypertrophous in thirteen of twenty-two cases of cerebral hæmorrhage, and in all of these the kidneys were diseased, being for the most part granular and atrophous. (Medical Times and Gazette, Nov. 24, 1855.)

[2] Addison, Guy's Hospital Reports, 1839, vi.

[3] Marcé, Schmidt's Jahrb., Nov. 1855.

When softening of the brain occurs in a *convulsive* form, its diagnosis is to be established by regard to the symptoms already described, p. 457. It is by a consideration of the prodromata and of the after-phenomena that the distinction may be made from epilepsy; it is by a similar process that tumour of the brain or of meninges may be diagnosticated.[1] That which is characteristic of softening is not the fact of the convulsion, nor the form which the convulsion takes, but the gradual development of intellectual, sensorial, and motor failure, such as has been described in the section upon symptoms; and the absence of those general and special changes which are characteristic of tubercular, carcinomatous, or other morbid growths.

The form of softening which is marked by *delirium* is not likely to be confounded with any other malady. It is essentially an affection of old age, and may be distinguished from simple senile congestion by the persistence of its symptoms, and by the speedy development of those signs of failure in nerve-power, to which so much attention has already been directed.

2. *Chronic softening,* when its course has been chronic throughout, has to be distinguished from tumour and from meningitis; and although the distinction is not possible in all cases, approximation to certainty may be attained in the majority by regard to the following considerations.

In *tumour* there is pain, intense in degree, subject to violent exacerbations, limited to and fixed in one locality; the special senses are affected, so that there is blindness or deafness, or the two combined, on one side generally, but sometimes on both; there are local paralyses and epileptoid convulsions; but, apart from the convulsions, unimpaired intelligence. There is often marked disturbance of the stomach, obstinate vomiting, and constipation; and there may be the signs of the tubercular, carcinomatous, aneurysmal, or syphilitic dyscrasiæ. In *chronic meningitis,* there is pain, generally distributed over the whole head, not very severe in degree, and, although varying in intensity, not subject to the paroxysmal exacerbations observed in tumours; there may be local paralyses, and these are especially observed in the muscles of the eyeball; there is much but intermittent mental excitement, and irritability of temper alternating with marked depression; there are disorderly spasms and paralyses of the limbs together with frequent but irregular accessions of fever; there is often a syphilitic taint, but there may be an entire absence of that dyscrasia, and the symptoms may have dated from a blow or fall.

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[1] See articles Epilepsy and Adventitious Products in the Brain.

In *chronic softening* there is dull headache, and gradual impairment of intelligence, motility, and sensibility, together with advancing years or a prematurely aged appearance, a feeble heart, rigid vessels, and most commonly some disease of old standing in such important viscera as the kidneys, heart, or liver.

Thus, to resume, the characteristic feature of tumour is pain, that of softening is failure of power, that of chronic meningitis is the mixture of excited with depressed functional activity. It is the progressive deterioration of cerebral faculty which marks out the disease we are considering; the patient begins to die, as it were, before his time, and his death begins in the highest element of his organism.

PROGNOSIS.—Occasionally there is complete recovery after an attack of acute softening; for example, apoplectic seizures have occurred, which have been followed by hemiplegia and mental dulness of many months' duration; these symptoms have passed away entirely, and upon post-mortem examination, after a number of years have elapsed, the signs of old softening have been distinctly discernible. It is possible, of course, that the softening may have originated in or around a "clot," but that possibility does not affect the general question of prognosis. Again there may be improvement, but not complete recovery; a patient may suffer a sudden apoplectic seizure, and may lie for days in a state of profound danger, the mind almost a blank, and the limbs hemiplegic; but after a time he may improve, and his improvement may continue for months, slowly going on from week to week; he becomes able to understand what is said, to speak or to make intelligible gestures, he may walk, or may even regain the use of his hand, and may remain more or less aphasic for an indefinite period. He may have a second attack, and one so characterised as to show that the other side of the brain has been affected, and he may be partially restored from this. At length a third or a fourth seizure comes, from which there is no recovery. Upon post-mortem examination in such cases distinct softenings may be found in such situations as to relate them to the first or second attack, and the cause of such softenings may be discovered in the obstructed arteries. The prognosis of softening, therefore, although unfavourable, is not necessarily fatal.

The prognosis is relatively favourable when the patient is young, and has been previously healthy; as, for example, when an apoplectic attack occurs during the course of rheumatic endocarditis in a young subject. It is unfavourable when the patient is old, or is affected by chronic disease of the kidneys, liver, or heart, when the arteries are rigid and the circulation low. It is favourable when the softening is, as it were, the result of accidental interference with the supply of blood; it is unfavourable when that interruption of the arterial circulation is but part of a general organic change.

The severity of an attack is to be judged of by regard to its mode of onset. If accompanied by either much congestion or by profound collapse, the symptoms, although very highly marked and widely distributed, may pass away; whereas the same amount of symptoms occurring without evidence of congestion or collapse would indicate, in direct proportion to their severity and extent, the gravity of the lesion. *Cæteris paribus,* the lesion is in proportion to the *extent* of the symptoms; and the prognosis is worse when the mind, sensation, and motion are all slightly impaired, than it is when either one of them alone is profoundly affected.

If the patient be young, and if there be no signs of general impairment of nutrition, hopes may be entertained that there will be some recovery of mental and motor power: but if the patient be old, or if there be weak circulation, and rigid vessels; and if the attack has had many forewarning symptoms, such as occasional forgetfulness, numbness of the extremities, and the like, the probability of restoration is very small, while the likelihood of increasing mischief or of renewed seizure is very great.

The prognosis of approaching death after an apoplectic or convulsive seizure is based upon the increasing rapidity and feebleness of pulse, the involuntary passage of the urine and fæces, and the general flaccidity of the limbs.

Treatment.—Attacks of softening may be postponed by attention to the following points:—1. The maintenance of an even temperature in the body: the feet and hands when chilly and blue should be put in hot water, or wrapped in and rubbed with warm flannels, and the patient should be placed in the recumbent posture, with the head only slightly raised. 2. The avoidance of long intervals between meals; food, easy of digestion, should be given frequently, and the patient, if old, should not be allowed to pass the night without nourishment. 3. The ready administration of some gentle stimulant when there is any tendency to occasional pallor or faintness. A glass of wine, or some sal-volatile and water, should always be at hand and should be given, not recklessly, but fearlessly if the premonitory symptoms become threatening. 4. Direction of the mental habits; easy and pleasant occupation of the mind, with careful abstinence from lazy inaction on the one hand, or violent excitement on the other. 5. Careful attention to the excretions, the skin, the kidneys, and the bowels. Exposure to cold is very prejudicial, and, although constipation and straining at stool are to be strenuously avoided, nothing is much more mischievous than the relaxation of close and too warm rooms, and the production, by medicines, of anything approximating purgation of the bowels.

When the premonitory symptoms are those of much headache and drowsiness, obvious relief may be gained by warmth to the extremities, and by the use of such diuretics as the liquor ammoniæ acetatis, with infusum scoparii, nitrate of potass, and spirits of nitric ether, or of juniper.

When there is the tendency to nocturnal delirium, a judicious administration of liquid nourishment, with very small quantities of wine, may suffice to give relief. Should this fail, the most useful medicine that I know of is the Indian hemp, in doses of a quarter to half a grain of the extract; and next in value is the chloral hydrate in doses of five or ten grains.

If there are marked symptoms of spasmodic or convulsive character, bromide of potassium in doses of from five to fifteen grains may be given three times daily with a bitter infusion and some diffusible stimulant, such as chloric or nitric ether.

On the occurrence of an attack, either apoplectic or convulsive, there is but little that can be done beyond the regulation of temperature and of secretion that has been already described; but when the attack has passed away something may be gained by the administration of cod-liver oil, hypophosphite of soda, and vegetable tonics. In many cases of chronic softening marked improvement follows the exhibition of cod-liver oil, and I am disposed to regard this as the most valuable agent in the treatment of the malady. It should be administered in conjunction with a fully nutritious but easily digestible diet, and with free exposure, without fatigue, to fresh air. It appears to me highly doubtful whether under any circumstances of softening of the brain the smallest good has followed either general or local blood-letting, the application of blisters, the administration of mercury or of iodide of potassium.

When recovery has advanced to a considerable degree, and some limbs remain paralysed, good has distinctly followed the exhibition of iron, and of strychnia in exceedingly small doses, and the cautious application of galvanism to the weakened muscles.

ADVENTITIOUS PRODUCTS IN THE BRAIN.

By J. Russell Reynolds, M.D., F.R.S., and
H. Charlton Bastian, M.D.,[1] F.R.S.

In this chapter is included a description of many diseases differing widely from one another in their pathological character, but agreeing in this, that they lead to the development within the cranium of some abnormal physical conditions, the nervous symptoms of which may bear close resemblance, *inter se*, during life.

Symptoms.—It has sometimes happened that, on post-mortem examination, tumours have been found in the brain, the existence of which had never been suspected during life. The patient may have presented no sign of cerebral disease, and may have died from an affection of the lungs or other organs; and, with the help conferred by post-mortem discovery upon the direction of questions as to past history, no evidence can be obtained of any symptom which can be referred to the brain. Such a case occurred to myself some years ago; several large hydatid growths being found in the brain of a young girl who had never suffered in any such way as to lead to the suspicion of cerebral mischief. These facts should be borne in mind when dealing with certain cases where signs of cerebral disease are present, but the diagnosis is obscure. When it is said that "a tumour cannot exist because of the absence of this or that symptom," it should be replied, that a tumour may exist without any symptoms at all.

There may be very highly marked symptoms, and yet these may be of such a character as to mislead. Dr. Abercrombie[2] has related examples of disease within the cranium, the locality and nature of which had been unsuspected during life, the patient's history having been such as to lead to a diagnosis of some affection of the stomach. Cases of the kind described by Dr. Abercrombie are rare, and it is probable that they will be rendered more so, as clinical examination becomes more minute. The fact, however, remains, that the complaint of a patient may be so marked with regard to dyspepsia, vomiting, constipation, and the like, and so trivial in respect of headache, giddiness, or other morbid sensations, that, although he has a tumour in his brain, no suspicion may be entertained of its existence, and the diagnosis that is arrived at may be malignant disease of the pylorus, or the liver.

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[1] The section on Morbid Anatomy is written by Dr. Bastian.

[2] Practical Researches, p. 337.

In other cases the symptoms of intracranial tumours are highly characteristic, and the diagnosis of their existence, their exact situation, and anatomical nature may be sometimes made with a precision and minuteness such as is scarcely attainable with regard to any other diseases.

The *intellectual* faculties may be unaffected; indeed, they very often are retained in their integrity when other functions of the brain are seriously impaired. The changes they present are of two kinds: there may be on the one hand great irritability of temper, a condition totally different from the previous habit of the individual, and with this, but very rarely, some mild delirium, or confusion of thought; on the other hand, there may be loss of memory, and general impairment of intelligence, with depression of spirits and listlessness. When convulsions occur, as they frequently do, during the few days that precede death, there may be profound coma as their sequel; but the convulsions which exist in the earlier period of the growth of tumours are often accompanied by only partial loss of consciousness, and are followed by none of the stupor which is so commonly observed in epilepsy.

Sensation is altered in various manners, but by far the most characteristic change is that of pain. Headache is often slight at the commencement, but afterwards it arrives at great severity; it is usually confined to a definite point or region of the head, and persists in that locality; it undergoes occasional exacerbation, and sometimes the suffering seems almost intolerable, and elicits from the patient agonizing cries; it is, however, rarely absent altogether during the intervals of paroxysm; it is increased by intellectual and physical exertion, by emotional disturbance, by sensational impressions, and by forced respiratory movements. It is sometimes almost the only symptom, but in rare cases it is absent altogether. The aggravation of pain which is occasioned by light, or noise, or movement, is such as to make a patient hold his head steadily between his hands, or bury it in the bed-clothes; and this intolerance has sometimes been regarded as hyperæsthesia, from which it differs widely. The sight and the hearing may be dull, but yet sensorial impressions intensify the headache. The sense of sight is often lost in one eye or in both; or there may be simply some mistiness or imperfection of vision, with dark or bright spots before the eyes. The iris does not often lose its irritability, but it is often found diminished; whereas, in some cases of complete blindness, it contracts readily on the admission of even a feeble light. Various changes may be observed in the ophthalmoscopic appearances, but that which is, perhaps, the most characteristic of tumour—although by no means constantly present—is enlargement of the veins, an enlargement sometimes to be observed in one eye only, sometimes in both. Hearing is less commonly impaired, but it may be lost completely on one side without having attracted the attention of the patient; noises in the ears are common, either with or without any diminution of the faculty. Numbness, tingling, creeping feelings, sensations of heat or cold, may exist in the limbs, or in certain tracts of skin over the trunk, and sometimes there may be distinct anæsthesia of parts. Vertigo is frequent, and often most distressing; usually it is relieved by closing the eyes and maintaining perfect rest; but sometimes it is aggravated by darkness, and the patient has to maintain a fixed gaze in order to ward off the feeling. Commonly the vertigo is of such a kind that the patient feels as if rolling over, or swimming along in space; and it is comparatively rare to hear complaint of the apparent rotation or motion of surrounding objects. Affections of the sight have been found most frequently when the tumour has occupied the anterior lobes of the brain, and least frequently when in the posterior lobes or cerebellum.

In the cases which have been placed on record, *convulsions* have occurred more frequently than *paralysis*, and among those which have presented the latter, one-half have exhibited the former. Convulsions, of epileptic form, often occur during the few days that precede death; but in certain kinds of tumour or of adventitious product in the brain, epileptoid convulsions may exist for years, and the cases presenting them may be termed "epileptic." When no general paroxysms occur, there are, very frequently, clonic spasms or tonic contractions of the muscles. As the result of an examination of a large number of cases, it may be stated that convulsions are most common when the disease is situated in the posterior lobes of the brain, or in the cerebellum, and least frequent when the anterior lobes are affected; the distribution being exactly the reverse of that which pertains in regard of amaurosis.[1]

Paralysis is sometimes observed in one muscle of one eyeball, such for example, as the external rectus, leading to convergent strabismus; or in all the muscles supplied by the third nerve on one side, so that ptosis and divergent strabismus, with dilated pupil, are the results. The speech may be also affected, the patient being unable to articulate certain sounds; the facial muscles may be so paralysed as to produce every degree of deformity; or the paralysis may be more widely distributed, and be hemiplegic, or, but more rarely, paraplegic in its form. Sometimes the lesion may be of such kind, and in such degree and locality, as to affect the nutrition of muscular and other textures. For example, in a case under my own care, where a tubercular mass involved the seventh nerve, and also the deep origin of the fifth, not only was there loss of sensibility on one side of the face, but the temporal muscle was much wasted, the conjunctiva and cornea became sloughy, and the mucous membrane of the mouth was aphthous and studded with vegetable parasites. The paralysis of cerebral tumour is developed—as a rule—slowly and insidiously; and when it occurs in one of the limbs, is sometimes preceded by pain or some other alteration of sensibility; but in a few cases it is produced as an "apoplectic" phenomenon, and may be the first symptom to attract the notice of either patient or physician.

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[1] Vide Auct., Diagnosis of Diseases of the Brain, &c. p. 186.

The *general* symptoms of tumour of the brain vary almost indefinitely, for they may be simply those of reflex or direct disturbance of the stomach and other viscera, when hydatid or fibrous growths are their producing cause; or they may be the special features of carcinoma, tubercle, or syphilis, when any one of those dyscrasiæ is the primary fact in the formation of the adventitious product. Thus they may, on the other hand, be all-important, and may partially conceal the cerebral disease, on the other they may be so trivial as to awaken no attention; while in an intermediate group they may have such character and relation as to render it possible to make an accurate diagnosis of the nature of the lesion. Thus, there may be tumours on the scalp, or, in other regions, glandular swellings of strumous character; or there may be distinct evidence of syphilitic deposit, of carcinoma, or of aneurismal dilatation of the vessels. Tuberculosis gives rise to the most common form of tumour in the child or young adult, and syphilis is the next in frequency in early life or middle age; while carcinoma is prevalent in direct proportion to advancing years. Sometimes there is obvious alteration in the general contour of the head, but such change is almost confined to the period of growth; whereas in later years there may be gradual prominence of one eyeball, or the distinct pressure of a growth through the bones. Under such circumstances the diagnosis is tolerably easy.

As a rule the commencement of symptoms is insidious, and their progress slow; but sometimes after a few premonitory phenomena there is a sudden attack of convulsions, or an apoplectic seizure. Under such circumstances a diagnosis is possible only by consideration of the subsequent history.

DIAGNOSIS.—In some cases it is impossible to gain even a hint of the nature of the malady, while in others the diagnosis is as certain as that of any disease with which we are acquainted. It will be convenient to speak first of the diagnosis of tumour generally, secondly of the diagnosis of the locality of growths, and thirdly of the recognition of particular forms of morbid product.

1. If convulsions be the prominent feature of the case the diagnosis is to be made from *epilepsy*, and here the distinction depends upon the recognition of symptoms over and above those of the latter disease. The convulsions are commonly epileptiform in type, but very often they present these differences,—they are irregular in development, there is not absolute loss of consciousness, there is little or no asphyxia, and no subsequent stupor, while the spasmodic movements are more marked on one side than on the other, they last for a longer time than is observed in epilepsy; and frequently terminate without being followed by anything like epileptic coma. In many cases the age at which they commence is so far advanced as to make epilepsy improbable, and there are symptoms—such as pain, affections

of the senses, and paralysis—which do not occur in simple epilepsy. Again, the mental state in a person the subject of tumour may remain quite intact, and may fail to present the peculiar sluggishness which is often, although by no means invariably, the concomitant of repeated epileptic seizures. Epilepsy is essentially a chronic disease, the commencement of which dates in a large proportion of instances from or soon after the period of puberty; it is characterised by fits of a peculiar type; and when uncomplicated it exists for years, and in the majority of cases without entailing any ulterior change in the functions of either brain or spinal cord. Tumour in the brain is of comparatively rapid development; it begins, as a rule,—to which there is the one exception of tubercular growth—after adult age has been reached, and most commonly when the period of middle life is passed; and when convulsions are present they are by no means the prominent symptoms of the malady, for, although often severe, they may be cast into the shade by the violence of pain and the loss of special senses.

It is possible that the early and even some of the advanced symptoms of tumour in the brain should be confounded with, or passed over as *hysteria.* Such mistake can only arise through carelessness, or through a prejudiced mode of dealing with the obscure affections of women. Not long since a lady consulted me, who was supposed to be hysterical, and who had been treated upon that supposition. Yet her symptoms had not commenced until after thirty years of age; she had violent paroxysmal headache, was blind of one eye, and deaf of one ear, and the amaurosis and the deafness had crept on slowly. The distinction from hysteria may be made by regard to age, affections of the special senses, the absence of the peculiar mental condition of the hysteric patient, and the nature of the paroxysms.

Chronic meningitis may be of such character that its physical conditions become identical with those of morbid growths, and its symptoms then pass into those of tumour. This is the case, for example, when there is syphilitic thickening of the membranes, which may at any time become so complicated by nodular thickening of either the membranes or the bones, as to give rise to the special conditions and symptoms of a tumour. Again, a tumour, of fibrous or carcinomatous character, the growth of which is habitually slow, may sometimes set up, in its neighbourhood, chronic meningitis, so that the symptoms of the two morbid processes may be found in association. Under either set of circumstances the diagnosis is possible by a regard to the mode of development of, and relative proportion between the symptoms. As already stated (see p. 487), the distinction between tumour and chronic meningitis lies here—that in the former the characteristic features are violent pain, marked diminution, or loss of one or more of the special senses, limited paralyses, integrity of mind, and occasional epileptoid convulsions; whereas in the latter the pain is slight, the special senses are perverted but not lost, the mind is damaged, and the convulsions are less distinctly epileptoid. In the former there is the predominance of pain, in the latter there is no such predominance, but a mixed condition of excited and diminished action in mind, sensation, and motility. In the case of chronic syphilitic meningitis the diagnosis may be assisted by the history of syphilis, or by the presence of its symptoms in a tertiary form. It must be remembered, however, that the diagnosis may often be carried to this point,—the obvious presence of chronic meningitis, and the probability of tumour; but this latter cannot be affirmed to exist because of the absence of its special symptoms (see page 491). On the other hand, in certain cases, there may be no doubt of the existence of a growth, when paroxysmal pain, &c. occur in a patient exhibiting the features of the carcinomatous cachexia.

From *chronic softening of the brain*, the diagnosis is to be made by recognising the absence of the characteristic features of that malady, viz. loss of mental power, of sensation, of motility. Certain cases of tumour may be taken for examples of chronic softening, and they are such as have been marked by a small amount of pain, by repeated convulsions, and gradual failure of intelligence. It would be impossible under such circumstances to make an accurate diagnosis.

2. The diagnosis of *the particular locality of a tumour* may sometimes be very minute and accurate, but in other cases vague, and not unfrequently erroneous. The side of the brain affected is usually, but not invariably, the same as that upon which the pain is felt, and on which the special senses and the muscles of the eyeball are affected; it is the opposite to that upon which spasm or paralysis occur in the limbs. Again, the locality of pain may be taken for a guide as to the situation of a tumour in the anterior, middle, or posterior lobes. Such guide, however, is not always trustworthy, for frontal pain may be the result of a cerebellar tumour. Upon analysing a large number of cases, I find that convulsions are most frequent in tumours of the cerebellum, and that they diminish in frequency as the seat of lesion advances forwards, *i.e.* through the posterior and middle to the anterior lobes of the cerebrum; and that amaurosis, impaired articulation, and intelligence observe a contrary relation to those lobes, being most common when the tumour is in the anterior cerebral lobes, and relatively less frequent as the seat of tumour retrogrades. Romberg has suggested that the position of a tumour on the upper surface or at the base of the brain might be determined by an observation of the effect produced on pain by forced inspiration or expiration: the pain of tumours, when seated at the base; being aggravated by inspiration, that of those on the upper surface by expiration, sneezing, or coughing. This Romberg explains by the rising and falling of the brain during the respiratory movements, and the consequent pressure of the mass against the upper or lower bony walls.[1] I have met with several cases which confirm Romberg's statement, but several others in which no reliance could be placed upon the test. The particular portion of the brain involved in a tumour may be sometimes determined by a careful consideration of the distribution of all the symptoms; such

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[1] Manual of the Nervous Diseases. Syd. Soc. Trans. vol. i. p. 159.

diagnosis, however, requires merely the application of anatomical and physiological knowledge, and needs no further notice here.

3. A diagnosis of *the nature of a tumour* is always of great importance both in regard of prognosis and of treatment. Sometimes all that can be accomplished is a guess, but sometimes tolerable certainty may be attained. Under certain circumstances we may distinguish between tubercle, syphilis, lead-poisoning, aneurism, hydatids, carcinoma, and hypertrophy.

Tubercle is the most common form of tumour in the child or young adult, and we might infer its presence if, in addition to a tubercular family history, there were the obvious features of the tubercular constitution, marked wasting of the body, together with an elevated temperature, and the presence of tubercular disease in the lungs or bronchial glands. It is somewhat curious to observe that the temperature of cases of cerebral tuberculosis is not raised to the degree, nor with the persistency, that is to be noticed in regard of tuberculosis elsewhere, but of all cerebral growths that which exhibits the greatest amount of elevation of temperature is tubercle. After puberty an examination of the chest is of great importance in the diagnosis, since it rarely happens that a healthy state of the lungs is found co-existent with tubercular disease of the cranium.

Syphilis may occur in such locality as to produce any of the varied symptoms which have been enumerated; but there are certain features which help to distinguish it from other forms of adventitious products in the nervous centres. Headache is rarely intense, but is prone to undergo nightly increase; actual loss of sight or hearing is not common; but implication of the third or sixth nerve is very frequently observed, so that patients exhibit ptosis, dilated pupil, and divergent strabismus, or more commonly inversion of the eyeball; there is often much depression of spirits, and further a wide extent of symptoms, the spinal cord as well as the encephalon being involved in the mischief. The characteristic features of this disease are, however, to be sought elsewhere, in the presence of periosteal thickening, eruptions on the skin, and such other phenomena as have been described in the article on Constitutional Syphilis.[1]

The *intoxication of lead* may be followed by such induration of brain substance as shall produce the symptoms of tumour; but the diagnosis turns upon a consideration of the previous history of the case, and the discovery of exposure to lead by trade, accident, or medicine: the previous occurrence of symptoms of lead colic; the presence of general cachæmia, of a blue line on the gums, and of lead in the secretions. The extensors of the hands and fingers are the most frequently paralysed; the extensors of the toes, the tibialis anticus, and peronæi are not rarely affected; the paralysed muscles become much impaired in their nutrition, and exhibit marked diminution, even extinction, of both contractility and sensibility on the application of powerful faradisation; but yet they may respond readily to a slowly interrupted battery-current of moderate power. It has been further observed that this loss of contractility to faradisation sometimes persists after there has been restoration of voluntary power.

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[1] Vol. I. p. 313.

Induration of the brain may occur in scorbutus, in rickets, or in epilepsy, but the diagnosis of its presence in either condition would be attended with much difficulty during life. Dr. Cohn observes that in one case, there was, in the instance of rickets, an entire want of intellectual disturbance;[1] whereas in epilepsy,—as described by Bouchet and Cazauvielh, and as observed by myself,—the presence of induration might be inferred from the progress of general intellectual decay; attention, apprehension, memory, and judgment failing; the patient becoming gradually incoherent, and general paralysis creeping on, while the fits increased in frequency but diminished in violence.

The *aneurismal* nature of a tumour could be guessed at only *per viam exclusionis,* or rendered probable by the observation of disease of similar kind in other portions of the arterial system; for it has been only in very rare cases that an aneurismal bruit has been discovered in the head during life; similar remarks may be made with regard to hydatid growths, and it must be remembered that hydatids in the brain may exist without producing any symptoms whatever.

The presence of *carcinoma* would be inferred only upon the recognition of the cancerous cachexia; and here age would be an important element in the consideration. Lancinating pains in the limbs are not, as Rostan supposed,[2] of any diagnostic value; but the diagnosis must rest upon the discovery of the carcinomatous dyscrasia, and the co-existence of tumours elsewhere, and especially of such as affect the integument and bones of the skull.

Hypertrophy of the brain, although not an adventitious product, is best described in this place. It may be met with in young children; but the only characteristic feature of the disease is enlargement of the cranium. Dr. West observes that this is "first apparent at the occiput, and the bulging of the hind-head continues throughout especially striking. The forehead may, in the course of time, become prominent and overhanging, but the eye remains deep sunk in the socket, for no changes take place in the orbitar plates, such as are produced by the pressure of fluid within the brain."[3] There is no prominence, but actual depression, of the fontanelles and sutures; the general nutrition of the child is imperfect, but there is nothing special in the cerebral symptoms which would lead to the diagnosis of this particular form of malady. Partial hypertrophy of the brain may be attended by no disturbance of the cerebral functions; the morbid condition, under such circumstances, can be recognised only by post-mortem examination.[1]

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[1] See Cohn, in Gunsb. Zeitschr. v. 35, 1854; Schmidt's Jahrb. Bd. 86, 1855, No. 6, p. 322; and Bouchet and Cazauvielh, De l'Epilepsie dans ses Relations, &c.

[2] Recherches sur le Ramollissement, p. 404.

[3] Lectures on Diseases of Infancy and Childhood, p. 9.

Morbid Anatomy.—In this place we do not profess to give an account of intracranial adventitious products as a whole, but shall strictly confine ourselves to such products or growths as have their seat in some part of the encephalon, and we must refer to another article for a description of the morbid growths which take origin from the meninges. Whilst it is indispensable, from a pathological point of view, to refer to these growths under different heads, it must not be supposed that we are able clinically to exercise the same precision. And neither, during the life of the individual, is it possible to determine whether a new formation, imagined to exist, has originated in the midst of the brain substance, or, having sprung up from one or other of the meninges, has merely grown into or pressed upon this secondarily. In both cases the symptoms produced may be almost identical. Similar effects are also, in rare instances, produced by the extension of an external morbid growth inwards through the orbit.[2]

In addition to the adventitious products more strictly so called, such as blood-clots, hydatids, &c., many varieties of tumours are met with in the encephalon. As in other parts of the body, these often present unmistakeable characters, though occasionally their histological composition is so indefinite as to make it extremely difficult to classify them. They exist either as distinct growths, with sharply defined outlines, or they may be, as it were, infiltrations passing insensibly, at their circumference, into the surrounding brain tissue. In the former case they are often enclosed in a fibrous envelope, of more or less thickness, which now and then may be found in a calcified condition; whilst the brain tissue around may be quite firm and healthy, or it may be softened to a variable extent. The softening may be simply white, or, if there have been much antecedent congestion, it may exhibit various shades of red; whilst in other cases the yellow gelatinous softening is met with, such as Rokitansky[3] and Cruveilhier[4] have described. This seems to occur most frequently around cancerous growths. At other times the brain tissue surrounding adventitious products is condensed and indurated.

Such adventitious bodies as silver and lead are met with only in minute quantities, and are for the most part diffused through the substance of the brain. Not being collected into distinct aggregations, we require the aid of the chemist to recognise their existence. Saline matters, also, either abnormal in kind or in quantity, may be diffused through the tissue of the nervous centres, when the nutrition of these fails and the brain matter is undergoing certain modes of degeneration. This is a subject upon which, at present, our knowledge is very defective.

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[1] See cases reported by Giacomo Sangalli, Gaz. Lom. 1858, quoted in Schmidt's Jahrb. Bd. 102. 1859, p. 22.

[2] Ch. Robin, Gaz. Méd. 1855, 6 et 13 Oct.

[3] Path. Anat. (Syd. Soc.), vol. iii. p. 419.

[4] Anat. Path., 8^{me} Livr. p. 5.

The various new formations and foreign bodies which are, from time to time, met with in the substance of the brain, may be thus arranged, though they will not all be treated of in the present article :—

1. Tubercle.
2. Syphilitic growths.
3. Cancer.
4. Melanotic tumours.
5. Gliomata.
6. Fibro-plastic tumours.
7. Fibrous do.
8. Osseous do.
9. Tumours of the pituitary and pineal bodies.
10. Nodules of grey matter on ependyma of ventricles.
11. Vascular erectile tumours.
12. Aneurisms.
13. Blood-clots.
14. Abscesses.
15. Plastic lymph on the walls of ventricles.
16. Cysts.
17. Calcareous and other concretions.
18. Entozoa
    - *a.* Cysticerci.
    - *b.* Hydatids.
19. Silver and lead.

It will be convenient to place also in the last section the remarks that we have to make upon hypertrophy of the brain.

1. *Tubercle.*—Tubercle in the brain is much more frequently encountered in children than in adults, but, as pointed out by Andral, it is not commonly met with in children under two years of age. The period of maximum frequency is thought to be from the third to the seventh year inclusive. It rarely occurs in the brain in children without at the same time existing in some other organ of the body. In 117 examinations of adults who had died of phthisis, Louis met with tubercle in the brain only in one case, whilst MM. Rilliet and Barthez discovered masses of tubercle in the brains of 37 out of 312 children in whom this morbid deposit existed in one or other of the remaining organs of the body.

Sometimes a single mass of tubercle exists in the brain, sometimes two or three, and in others a large number of smaller masses. In size the separate masses vary between that of a millet seed and a large hen's egg—those most commonly met with, however, being about equal to a filbert or a small walnut. They are mostly spherical in form, but are occasionally more or less lobulated. All parts of the brain, from the surface to the centre, are occasionally the seat of this deposit. Perhaps, having regard to its size, the cerebellum is the most favourite seat of tubercle. It is so regarded by Dr. Wilks and by Sir William Jenner; and Andral was, doubtless, of the same opinion, since, without regard to size, he placed the cerebellum second, after the cerebral hemispheres, in the order of frequency of site. After these, in order of frequency, Andral names the pons Varolii, the medulla oblongata, the spinal cord, the peduncles of the cerebrum and cerebellum, the optic thalamus, and the corpus striatum. Very frequently, when masses of tubercle are situated in or upon the cerebellum, they impede by their pressure the return of blood through the venæ magnæ Galeni, or the straight sinus, and so cause effusion of serum into the ventricles, and all the symptoms of chronic hydrocephalus.[1] Very rarely, almost the whole of the cerebellum, or one of its lobes, may be, as it were, replaced by tubercular matter.[2]

The usual condition in which tubercle is met with in the brain is, according to Rokitansky, in the form of masses in the size and shape already mentioned, "of a yellow or yellowish-green colour, of the consistence of lard or cheese, and firm, but easily lacerable." It is remarkable that the grey, translucent form of tubercle is rarely, if ever, met with in the brain. Lebert says he has seen it rarely, and Rokitansky believes "that there are some rare cases which prove that tubercle in the brain does, in part at least, commence in the grey translucent form, for portions of a tubercular mass are sometimes found in that state." He believes that it rapidly passes over from this form into that of the yellow cheesy tubercle. So far he is very much in accord with Virchow,[3] who holds that each mass of cerebral tubercle is in reality made up by the aggregation of a multitude of small miliary tubercles. Each mass is formed, not by the growth of one original focus, but "by the continual formation and adjunction of new foci at its circumference." He adds:—"If we examine one of these perfectly yellow, or white, dry, cheesy tubera, we find immediately surrounding it a soft, vascular layer which marks it off from the adjoining cerebral substance—a closely investing areola of connective tissues and vessels." In this layer the young granules[4] are formed. They are continually produced at the circumference, "and the large tuber grows by the continual apposition of new granules (tubercles), of which every one singly becomes cheesy."[5] But though this is the condition in which growing masses of tubercle are met with in the brain, at a later stage the process of increase stops, and, owing to an irritative process, a fibrous envelope gradually forms round the mass, so as completely to isolate it from the surrounding brain tissue. This condition is so general that many pathologists have held that all tubercles occur in an encysted condition in the brain.[6] The thickness of the fibrous envelope varies with age—it may be an almost imperceptible layer of fibrous tissue, or it may attain a remarkable thickness and almost cartilaginous consistence. Occasionally, even, it becomes completely calcified.[7] Softening is met with, at times, in the centre of the tubercular masses; and, very rarely, in encysted tubercle the whole contents may undergo this change. Such a case is reported by Dr. Ogle,[1] in which a cyst the size of a pigeon's egg, with thin and friable parietes, was found in the pons Varolii, containing a "yellow glairy fluid in which a number of light-coloured soft particles of albuminous matter existed." More rarely still, in the brain, the tubercular mass has been found to have undergone a process of cretification.

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[1] Wilks' Path. Anat. 1859, p. 158. This fact is strongly insisted upon, also, by Sir W. Jenner in his clinical teaching.

[2] Vide Hooper's Morb. Anat. of the Human Brain, p. 60, pl. xi.

[3] Cell. Pathol., transl. by Chance, p. 477.

[4] Grey granulations.

[5] Although differing so widely in their views as to the nature of tubercle, Rokitansky's description of the circumference of these masses in the brain is almost identical. He says:—"An extremely moist and jelly-like cellular structure connects the tubercle with the surrounding cerebral tissue. * * * This stratum further contains, scattered mostly through its inner part, some small grey or greyish yellow tubercles, which occasionally unite with the great central mass."—Loc. cit., vol. iii. p. 429.

[6] Vide Andral's Précis d'Anat. Pathol. t. iii. p. 841.

[7] Dr. Ogle, Brit. and For. Rev., Oct. 1864, p. 463.

The brain tissue around masses of tubercle is often perfectly natural, at other times it may be congested, more or less softened, or even indurated.

2. *Syphilitic Growths.*—These are very rarely met with in the substance of the brain. Instances have, however, been recorded. Dr. Aitken saw a "gummatous tumour" occupying the left optic thalamus, in a patient of Dr. Goodfellow's, who had suffered from syphilis, and some of whose children had died from inherited secondary syphilitic lesions.

Dr. Wilks has never seen independent tumours of this kind in the cerebral substance, though he believes, from the symptoms observed in some cases, that such deposits were very likely to have existed. A firm, tough, yellowish lymph-like mass, of syphilitic origin, has frequently been met with intervening between, and connecting the dura mater with the brain. And although it seems most probable that the primary seat of this is the dura mater, still it is desirable to mention it here on account of the serious way in which the brain matter is often implicated. Dr. Wilks records [2] a characteristic example of this kind of lesion, met with in the post-mortem examination of a woman of low character, who was believed to have suffered from syphilis. He says: "At the anterior fossa the dura mater was united to the bone by a firm, yellow lymph; here also the bone was slightly roughened, but not carious. The dura mater on the inner side was firmly and inextricably united to the anterior lobes of the brain, especially on the right side, and corresponding to the anterior fossa of the skull. On attempting to separate them, a quantity of hard yellow material was seen uniting them together. This filled up the sulci, and involved the cineritious substance. On the right side it had penetrated to the medullary matter, and here the adventitious substance formed a tumour, tolerably circumscribed on its deep side, the size of a walnut." In the liver were some of the characteristic tough masses, corresponding with a puckered and cicatriform condition of the surface above them.

3. *Cancer.*—Cancerous growths in the brain are, according to Lebert, decidedly more frequent in the second half of life, though they are met with occasionally in youth, or even in childhood. Dr. Walshe also found that out of 56 persons affected with cerebral cancer, 26 died between the ages of 40 and 60 inclusive, whilst 5 died before the 10th year, and 5 between the 10th and 20th years. In about one-half of the total number of cases cancer of the brain is primary.

All three forms of cancer may occur in the brain, though encephaloid is by far the most common: next to this Lebert speaks of a lardaceous intermediate kind. It may exist either in the form of a distinct tumour, or it may infiltrate parts of the brain. The growths are usually solitary, though occasionally two or even more may be met with. The size of the cancerous mass varies from that of a pea up to an orange, or even larger. Occasionally the greater part of one hemisphere may be implicated. The cancerous mass is very rarely enclosed in a sort of fibrous cyst, but in the majority of cases it passes, at some part of its periphery, almost insensibly into the adjacent brain tissue. The colour is occasionally the same as that of brain tissue, though various tints of rose, yellow, and even green may be met with either singly or intermixed: very many cancerous growths in the brain are said to have a yellow colour. All parts of the brain are liable to be affected. Cancerous tumours are frequently found imbedded in the midst of the hemispheres, and, according to Lebert, those near the convexity usually attain the largest size, whilst those in the pons and medulla are usually the smallest, owing to the more rapid death of the patient when the growth occurs in these situations. The duration of life varies considerably; thus in 6 out of 11 cases inquired into by Lebert, the growth seemed to have proved fatal in about 6 months, whilst in 4 the symptoms extended over a period of from 2 to 5 years.

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[1] Trans. of Path. Soc. vol. v. p. 26.

[2] Guy's Hosp. Rep. 1863, p. 49.

The consistence and amount of vascularity of the cancerous growth vary much in different cases. Effusions of blood may be met with in the midst of soft cancerous masses, and not unfrequently cysts are developed in their interior, which contain a thick glairy fluid. The surrounding brain tissue may be natural (which is frequently the case), or it may be softened, or, still more rarely, in a state of induration. The softening may be white, red, or of the yellow[1] variety.

Lebert records one instance of a cerebral cancer which, by its progressive growth, caused a large perforation of the skull, in the situation of the coronal suture.

4. *Melanotic Tumours.*—These are found in the shape of small nodules, generally varying in size from that of a pea to a bean. They may exist in the deeper parts of the brain, or at its surface, in the grey matter of the convolutions.[2] Sometimes these growths may be cancerous in their nature, but others are certainly not so. The black colour is due to the infiltration of the cells of the growth with black granular pigment, similar to that met with in the choroid coat of the eye. Dr. Clendinning[3] found a mass of melanoid deposit in the upper part of the right corpus striatum, as large as a horse-bean, and, external to this, a hard pea-sized mass. Similar new formations existed in the centrum ovale, and in the right lobe of the cerebellum. In this individual, growths of the same kind existed also, in great numbers, in the subcutaneous tissue, and in most of the internal organs except the lungs.

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[1] Dr. Ogle, in Journal of Mental Science, 1864, p. 229, cases 1 and 4.

[2] Hooper's Morb. Anat. of Human Brain, 1828, pl. xii. figs. 2 and 3.

[3] Trans. of Path. Soc., vol. i. p. 42.

5. *Gliomata.*—These growths, to which the above name has been given by Virchow, take their origin in the neuroglia or interstitial connective tissue of the brain. They are, in fact, formed by a localised hyperplasia of the neuroglia, and contain no nerve elements in their composition. These tumours are never sharply defined from the surrounding brain tissue, to which they bear a certain superficial resemblance. On section, however, they are often seen to have a somewhat translucent, bluish-white appearance, whilst at the same time they may be firmer and rather more vascular than the brain tissue itself. Gliomata are usually solitary, and of slow growth, so that they may exist for a long time without producing any very appreciable symptoms. They often attain to a considerable size—that of an orange, for instance, and occasionally they are even much larger than this. They are most frequently met with in one or other of the posterior cerebral lobes, and after this, perhaps, they occur on some part of the upper and outer portion of the cerebral hemispheres oftener than in other situations. The tumours are composed of an intercellular substance, which varies in quality and consistence in different parts of the brain, and of an abundant mixture of cells and nuclei. The cells are variable in shape and size—the smaller ones occasionally possessing fine prolongations which are continuous with those of adjoining cells. There are two principal varieties of gliomata: the soft and the hard. The former, containing a soft basis substance, and numerous moderately large cells, are closely allied to medullary sarcomata; whilst the latter, having a harder and firmer basis substance, and small cells with highly refractive nuclei, have close affinities to fibrous tumours.

Dr. Cayley[1] has recorded an interesting case in which a tumour of this kind, about the size of a large walnut, involved all the deeper parts of the right side of the pons, the right processus ad testes, the corpora quadrigemina to some extent, the right half of the valve of Vieussens, and the fibres of origin of the right fourth and fifth nerves.

6. *Fibro-plastic Tumours.*—These growths most commonly arise from the pia mater; still occasionally they take their origin in the substance of the brain itself. They have been found in the midst of the hemispheres, in the pons, and in the cerebral peduncles. They vary in size from a pea to a hen's egg, are mostly spherical or ovoid in shape, and with a surface which is often mammilated or slightly lobulated. Their colour is generally rose-red, mixed with yellowish and even greenish tints. The amount of vascularity differs in different tumours, and in different parts of the same growth. They contract no adhesions, and, in general, do not infiltrate neighbouring parts, although they erode by their growth and consequent pressure.

Dr. Bristowe has recorded[2] a characteristic instance of the occurrence of a tumour of this kind in a man aged 33 years. The growth was irregularly spherical, and about one square inch in bulk. It arose from the right half of the pons and from the corresponding crus cerebelli, whence it extended for a certain distance into the medulla oblongata. The surface was lobulated, and had somewhat the appearance of brain substance, owing to its colour and the arrangement of vessels on its surface. There were no defined limits to the deeper portion of the tumour, which passed insensibly into the surrounding brain tissue. On section, the substance of the growth was greyish and slightly translucent, interspersed with patches in which the vascularity was more marked than it was elsewhere.

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[1] Trans. of Path. Soc., vol. xvi. 1865, p. 23.

[2] Ibid. vol. vii. p. 28.

Dr. Ogle [1] has reported a case of fibro-plastic infiltration, in which the new product, instead of forming a distinct tumour, had infiltrated itself into the tissue of the left optic thalamus, so as to make this body almost twice its natural size.

7. *Fibrous Tumours.*—These growths are very rare, and comparatively few cases are on record. They are mostly small and spherical, varying in size between that of a small pea and a walnut. M. Reignier[2] found a pedunculated growth of this kind of the size of a large filbert, growing from the valve of Vieussens; and in the Trans. of the Path. Soc., vol. v. p. 18, an account is given of a fibrous tumour about the same size, which was found projecting into the left lateral ventricle, from the side of the corpus striatum. Lebert records two cases: in one a tumour of the size of a pea was found in the pons, composed of a firm, elastic, yellow, and somewhat gelatinous tissue, but presenting the usual microscopic characters; in the other, 17 fibrous tumours were situated upon the ependyma of the lateral ventricles, varying in size between a pea and a small cherry stone, and of a white or slightly yellowish, or even rose colour in some places. On section they were homogeneous, and somewhat translucent. Several tumours were situated on the septum, and superficially they were all covered by epithelial cells, similar to those of the ependyma. The substance of the brain, in other respects, was apparently quite healthy, and there was no excess of fluid in the ventricles.[3]

8. *Osseous Tumours.*—True bony growths in the substance of the brain are extremely rare, still they have been met with. Dr. Bristowe[4] found a growth of this kind occupying the position of the infundibulum and corpora albicantia. It was a hard conical mass about as large as a horse-bean, whose apex rested on the pituitary body, and whose base assisted in forming the floor of the third ventricle. It "was wholly unconnected with the dura mater or osseous parietes of the skull." On microscopic examination, it presented the characters of true osseous tissue, with perfect lacunæ and canaliculi.

9. *Tumours of the Pituitary and Pineal Bodies.*—Both these bodies are occasionally found in a morbid condition, and more or less enlarged. Cysts are then frequently met with in their interior.

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[1] Trans. of Path. Soc., vol. vii. p. 12, pl. ii.

[2] Bullet. de la Soc. Anat., t. ix. p. 120.

[3] Anat. Path., vol. ii. p. 71.

[4] Trans. of Path. Soc., vol. vi. p. 25.

a. Pituitary Body.—Lebert considers the enlargement of this body to be a kind of hypertrophy. Several cases are on record. In one of them related by Rayer,[1] this body was about 1½″ in diameter, whilst its tissue was also more dense and resistant than natural. Vieussens[2] found the "gland" as large as a hen's egg, soft, and containing in its interior a greyish-white glutinous fluid. Heslop[3] records a remarkable case in which the tumour was soft, deep grey, and of the size of a large walnut, containing a small cavity with fluid in its interior. It occupied the region of the pituitary body, and also extended posteriorly as far back as the pons, and antero-laterally to the fissures of Silvius, so as to occupy the whole interpeduncular space. The corpora quadrigemina were flattened antero-posteriorly, from pressure. Abercrombie[4] also refers to a case described by Dr. Powell, in which there was found "a tumour, the size of a hen's egg, containing a thick purulent fluid under the anterior part of the brain, and interposed betwixt the optic nerves, which were much separated by it from each other. Below it was attached to the pituitary gland, which was very soft, and enlarged to five or six times its natural size." Davaine[5] records three cases in which small cysts (supposed to be hydatids) were found in the pituitary body.

b. Pineal Body.—Hooper[6] says, speaking of this body:—"It is sometimes converted into a *cyst*, the whole of the natural structure being destroyed. This cyst is firm and membranous, and I have seen it of the size of a tamarind stone. The contents of one which I examined were, a turbid serous fluid, with small particles of solid albumen." Dr. Ogle[7] also relates a case in which "the brain and membranes were natural, excepting that the pineal gland was exceedingly enlarged and very adherent, posteriorly, and contained two cavities, each full of transparent fluid, situated immediately below its investing membrane."

10. *Formation of Grey Matter upon the Ependyma of the Ventricles.*—Rokitansky and Virchow[8] have both described the existence of cerebral grey matter upon the internal surface of the lateral ventricles, in situations where, naturally, grey matter does not exist. It occurs in the form of numerous small tubercles, from the size of a mustard seed to that of a cherry stone.

11. *Vascular erectile Tumours.*—These growths are very rare. Nevertheless, Lebert has given the particulars of five cases in which they were found.[9] In all they were discovered *post mortem*, but had

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[1] Archiv. Gén. de Méd., 1re Sér. 1823, t. iii. p. 350.

[2] Nov. Vasor. Corp. humani Syst.; Amstelodami, 1705, p. 248.

[3] Dublin Quarterly Journal of Medicine, Nov. 1848.

[4] Diseases of the Brain, &c. 3rd ed. 1836, p. 438.

[5] Traité des Entozoaires. Paris, 1860, p. 656.

[6] Morb. Anat. of Human Brain, 1828, p. 43, pl. xii. fig. 8.

[7] Brit. and For. Rev., July 1865, p. 235. He also adds that the Museums of King's College and of St. Thomas's Hospitals contain one specimen each of an enlarged pineal gland, hollowed out into a cyst.

[8] Würtzburger Verhandlungen, t. ii. p. 167.

[9] Guérard, Bullet. de la Soc. Anat., t. viii. p. 223.

given rise to no notable symptoms during life. In one of these cases the growth was lodged in the right lateral ventricle, and was a development from the choroid plexus, but in the other four the tumours were imbedded in the cerebral substance.

In the case related by Farre[1] the growths were multiple; two of the same size being met with in the medullary substance of the left hemisphere, and several small growths in the corpora striata and cerebellum. Lebert[2] himself has minutely described an erectile tumour, of the size of a hen's egg, found in the posterior lobe of the right cerebral hemisphere, and Luschka[3] met with one of the same size in the left anterior cerebral lobe, which was surrounded by softened brain substance. Lastly, Förster[4] alludes briefly to an erectile tumour of the size of a nut, found in the grey cortical substance of one of the hemispheres, the cavernous spaces of which were found to communicate with neighbouring dilated veins. The tumours described by Lebert, Farre, and Guérard were made up almost entirely of fine vascular ramifications.

12. *Aneurisms.*—The intracranial aneurisms which are best known are those occurring on some one or other of the larger arterial trunks at the base of the brain, or on some of the branches of the circle of Willis lying in the midst of the pia mater, and therefore whilst they are still on the surface of the proper brain substance. Such aneurisms belong to the meninges, and will not be further referred to in this place.

There are, however, aneurisms belonging to the encephalon itself, whose existence has only recently been discovered, and which are remarkable principally for their small size, and on account of their frequent numerical abundance within the same brain. These were first detected and examined by MM. Charcot and Bouchard,[5] the latter of whom has shown, not only their frequent and close association with the phenomena of intracranial hæmorrhage, but also their apparent dependence upon a certain general pathological condition of the small encephalic arteries, which may exist alone, or may be associated, in various degrees, with the more familiar atheromatous degeneration. The pathological condition of the arteries favourable to the formation of these minute aneurysms is one of fibroid degeneration—a process of *sclerosis* in which there is brought about a great increase in the number of connective tissue nuclei on the perivascular sheaths, and also in the walls of the vessels, whilst the muscular fibre cells of the middle coat are gradually replaced by fibrous tissue. This change diminishes, or even destroys, the elasticity of the arterial coats, so that when, from any increase of the ordinary blood pressure, they have once become unduly dilated, they are unable to regain their normal calibre. In this way, by the incidence of increased pressure upon degenerated parts, are produced the various kinds of aneurismal dilatations, whose characters differ according to the degree and extent of the morbid changes in the parts involved. Thus, we may have uniform dilatation of an arterial branchlet, for a certain portion of its length, or this uniform dilatation may be interrupted by constrictions at intervals, owing to the presence of similarly situated sounder portions of the arterial walls. The kind of alteration with which we are more particularly concerned at present, however, and which is also the most frequent, is due to an altogether local and circumscribed change, and results in the formation of the minute and more or less spherical *miliary aneurisms*, as MM. Charcot and Bouchard propose to name them.[1]

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[1] Leubuscher, Die Patholog. und Therap. der Gehirnkrankheiten, p. 413, Berlin. Original reference not ascertained.

[2] Anat. Path., t. i. p. 213.

[3] Archiv für Path. Anat. t. vi. 1854, p. 458.

[4] Lehrb. der Patholog. Anat. p. 418.

[5] Bouchard, De la Pathologie des Hémorrhagies cérébrales. Paris, 1867. See also joint papers by MM. Charcot and Bouchard, in the Journal de Physiologie, 1868-69.

These miliary aneurisms are very rarely met with before the middle of life, and are most common in the very aged. They are visible to the naked eye, and can be seen readily with the aid of a pocket lens. Their diameter varies between $\frac{1}{125}''$ and $\frac{1}{25}''$, and they are attached to vessels which seldom exceed $\frac{1}{50}''$ in diameter. Sometimes only two or three can be detected in the same brain, though more frequently they exist in much larger numbers. Bouchard has found even more than one hundred in the same brain. They may be met with in all parts of the encephalon, though with different degrees of frequency in different situations. Hitherto they have been found most frequently in the optic thalami, and then, in decreasing order, in the pons Varolii, the cerebral convolutions, the corpora striata, the cerebellum, the medulla oblongata, the middle cerebral peduncles, and, lastly, in the white matter of the cerebral hemispheres. When abundant in the convolutional grey matter, a number of minute and variously coloured spots may be seen, after the membranes have been stripped off, lying exposed on the surface of the convolutions; whilst, when sections are made, others may be recognised in the deeper strata of the grey matter. Whether occurring in this situation or in the more central parts of the brain, the colour of the minute aneurismal grains varies from a bright red or violet, to a yellowish or even black hue;[2] according as they contain in their interior normal fluid blood, or more or less altered blood pigment. Occasionally the aneurisms seem to undergo a natural process of cure. Their wall, as well as that of the enveloping and sometimes adherent lymphatic sheath, becomes thickened by an increased growth of connective tissue elements, whilst at the same time the white corpuscles of the blood have a tendency to adhere to their inner surface. The fibroid change creeps on, bringing about, sooner or later, a union between the wall of the aneurism and its sheath, the gradual thickening of these, and an extension of growth inwards, probably owing to a further organization taking place in the substance of the adherent and fused white corpuscles. Thus may the cavity of the aneurism be gradually diminished, till at last this, and even the minute vessels on which it is situated, may undergo complete obliteration.

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[1] Cruveilhier (Anat. Patholog., Liv. xxxiii. pl. ii. fig. 3) figures and gives an accurate description, so far as it goes, of these very miliary aneurisms under the name of "apoplexie capillaire à foyers miliaires." He was therefore ignorant of their real nature, though perfectly familiar with the naked-eye appearance.

[2] Occasionally this blood pigment, in the form of amorphous yellow grains mixed with hæmatoidine crystals, may be principally collected around one of these aneurisms (which has been ruptured), though within its enveloping lymphatic sheath.

Such intra-cerebral 'miliary' aneurisms may co-exist with other and much larger aneurisms of the vessels of the pia mater. Bouchard speaks of a case of this kind where the aneurisms of the arteries of the pia mater were not only exceedingly numerous, but varied in size between a pea and a cherry-stone. In other cases, minute aneurisms of these meningeal arteries may be met with precisely similar to those co-existing on the vessels in the midst of the brain substance. Frequently when miliary aneurisms are met with in the smaller cerebral arteries, the larger arteries at the base of the brain exhibit marked atheromatous changes: such a coincidence, however, is by no means invariable.

Although the majority of intra-encephalic aneurisms are minute, and such as we have described, occasionally others of larger dimensions are met with. The size of the largest, however, could rarely exceed that of a small walnut, seeing that the arteries of the brain are comparatively small before they leave the pia mater to penetrate into its substance. We have collected the records of five cases of this kind of aneurism, and in each of these the patient's death was occasioned by the rupture of the sac. The first is related by Dr. Crisp,[1] and in this a boy aged fourteen died from the rupture of one of two small aneurisms on the anterior cerebral artery, in the substance of the anterior lobe. The aneurism which burst was as large as a horse bean, whilst the other was about the size of a pea, and was filled with laminated fibrin. The next case was recorded by Dr. Van der Byl,[2] and was altogether remarkable from the fact that the aneurism, which was situated on the left posterior cerebral artery in the substance of the brain, was as large as a hen's egg, and was almost filled with laminated fibrin. In one case recorded by Dr. Gull,[3] a small pyriform aneurism, "having much the appearance and size of a withered grain of wheat," burst in the centre of the pons Varolii, and was found in the midst of a coagulum weighing two drachms. Dr. Gull gives the details of another case in which an aneurism about the size of a small filbert, situated on the middle cerebral artery, in the anterior part of the middle cerebral lobe, was found in a girl aged seventeen, surrounded by a large recent coagulum, and by softened brain tissue. The other arteries of the brain are said to have been healthy. Lastly, the writer has himself recorded a case[4] in which an aneurism, about ⅓″ in length and of an elongated pyriform shape, with a distinct rupture in its larger extremity, was taken from the midst of an enormous effusion of blood into the outer part of the right corpus striatum and adjacent portions of the hemisphere. In this case there was an atheromatous condition of the arteries at the base of the brain, and, besides the larger aneurism, four or five of the small miliary aneurisms were found in different parts of the organ.

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[1] Diseases of Arteries, p. 165.
[2] Trans. of Path. Soc., vol. vii. p. 129.
[3] Guy's Hosp. Reports, 3d Series, vol. v. (1859), p. 297.
[4] Trans. of Path. Soc., vol. xviii. 1867.

13. *Bloodclots.* See Art. "Apoplexy."

14. *Abscesses.* See Art. "Abscess in Brain."

15. *Plastic Lymph on the surface of the Ventricles.*—A well-marked instance of this has been related by Dr. Wilks.[1] It occurred in a man who had fractured the left orbital plate of his frontal bone by a fall. A portion of the broken bone had torn through the dura mater and had injured the anterior lobe of the brain, so as to lead to the subsequent production of an abscess in this situation. The man died after seventeen days: "on incising the roof of the ventricle (left) a membrane was found within it; and on cutting this through it was found to consist of a layer of lymph, which completely lined the cavity. Some purulent matter escaped from within it. It covered the roof, the floor, and extended from the anterior to the descending cornu, and was so tough that it was capable of being removed entire; it formed, indeed, a complete cast of the cavity, and resembled a croupous membrane, as seen on the trachea in inflammation of that organ. The surface of the ventricle was soft, and in parts tore when the membrane was removed; but in most places it could be cleanly taken off. The foramen of Munro was closed, and the right ventricle contained only some turbid serum." It was uncertain whether there was a communication between the abscess and the ventricle, but the lymph was undoubtedly produced on the surface of the latter.

16. *Cysts.*—It seems extremely doubtful whether simple serous cysts are ever met with in the substance of the unaltered brain tissue. Those most likely to be of this nature are small cysts from the size of a pin's head to that of a mustard seed, which are sometimes met with beneath or projecting above the surface of the lateral ventricles. They occur either singly or in groups.

The corpora striata, on section, sometimes present the appearance of small cysts, even as large as a pea. These may, however, be either sections of dilated lymphatic canals, or cavities left after minute softenings.

It is true that larger cysts are not unfrequently met with in the brain, but these, when not due to one of the two forms of cystic entozoa, to be hereafter described, should rather be termed pseudo-cysts, since they are not primary formations, but have, in all probability, resulted from the modification of pre-existing pathological states. Such cavities or pseudo-cysts mostly result from the later changes taking place in the seat of old effusions of blood,[2] or of circumscribed softenings; or else they are due to the softening of encysted tubercular[1] or cancerous nodules.

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[1] Trans. of Path. Soc., vol. xv. p. 5.

[2] Many cases are recorded by Dr. Ogle (Med.-Chir. Rev., July 1865, p. 212).

Cysts occasionally form in the substance of cancerous growths in the brain, and, as before stated, they have several times been met with in the interior of enlarged pituitary and pineal bodies.

17. *Calcareous and other Concretions.*—These are not unfrequently met with in the brain, and are mostly due to changes which have taken place in masses of tubercle or in old abscesses. Such concretions consist for the most part of phosphate and carbonate of lime, and only contain a small quantity of animal matter. More rarely concretions may be met with which seem to have resulted from previous effusions of blood: thus Lassaigne[2] analysed a mass which was found to be composed almost wholly of fibrine, and contained only four per cent. of phosphate and carbonate of lime, with traces of cholesterine.

Concretions known as "brain sand" are very common on or in the pineal gland and its peduncles.

18. *Entozoa.*—Two kinds of parasites only have been met with in the human brain, and these always in an immature or larval condition. They are the *Cysticercus*, and the *Hydatid* or *Echinococcus cyst:* the first representing the second, or *scolex* stage, in the development of *Tænia Solium*, and the other an equivalent stage of *T. Echinococcus*, an animal which exists abundantly in its mature condition in the alimentary canal of dogs. Goeze and Zeder[3] have recorded two cases in which they suppose the *Cœnurus cerebralis* to have been met with, but they have not been supported by other observers, and Davaine believes these cases, in reality, to have been instances of hydatid disease. Certainly, as he says, the descriptions these writers have given are obscure and inexact, and we may well imagine mistakes to have been made, when we consider what was the state of knowledge concerning helminths at the time in which they wrote.

a. Cysticerci.—In the brain these vary in size from that of a pea to a small horse-bean, or even larger. The serous cysts, in which they are usually enclosed in other situations, are often absent entirely, so that they are bounded only by a smooth layer of unaltered or somewhat compressed brain substance. They often exist in large numbers in the same brain, and are very rarely solitary. From ten to twenty are frequently met with. Cruveilhier[4] records an instance in which more than 100 were found within the cranium of the same individual, and of these about 50 were lodged in the cerebellum. They may be found in almost all parts of the brain, but, speaking generally, they are by far the most abundant at the surface of the brain, in, or in close connexion with, the grey matter of the convolutions. They are extremely frequent in the pia mater, also where they press upon and partially imbed themselves in the surface grey matter. Sometimes they are lodged in the midst of the grey matter itself, whilst more frequently still they are found intervening between this and the white substance. They have, moreover, been seen in the midst of the white substance, in the central ganglia, in the pons, in the crura cerebri, and in the cerebellum as before stated; whilst Cruveilhier says he has seen real cysticerci in the choroid plexuses of the lateral ventricles. In the latter situation they have to be carefully discriminated from the small, non-parasitic cysts which so frequently occur in the same locality.

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[1] Trans. of Path. Soc., vol. v. p. 26.

[2] Clinique Méd., t. v. p. 8.

[3] Nachtrag zur Naturgesch. der Eingeweidewurmer, 1800, pp. 308 and 313, tab. ii. figs. 5–7.

[4] Anat. Path. Gén., t. ii. p. 88. Paris, 1852.

Although usually giving rise to but slight changes in the surrounding brain matter, the cysticerci themselves undergo important modifications with age. It is desirable that this fact should be known, in order that pathologists may recognise them in their different stages, and that individual developmental modifications may not be mistaken for specific distinctions. According to Davaine,[1] "Les altérations portent, d'une part, sur la vésicule qui est devenue plus ou moins globuleuse, plus volumineuse, sans jamais cependant avoir acquis un grand volume, irregulière, quelquefois divisée en lobules ou même double; d'une autre part elles portent sur la tête dont le rostre et les ventouses sont envahis par une matière noirâtre, pigmentaire. Les crochets sont recouverts à leur base par cette matière. Dans une période plus avancée on les trouve en désordre, diminués de nombres ou même ils ont disparu. L'ouverture de la vésicule rétrécie ou oblitérée ne laisse plus sortir le corps; la tête invaginée dans celui-ci ne peut non plus en être extraite par une pression ménagée; sa présence ne peut être reconnue que par la dilacération des parties." It should also be added that in those cases where the cysticercus is non-encysted—as when it is lodged freely in one or other of the ventricles—it tends to grow more easily into the form of a tape-worm, by the elongation and segmentation of the neck of the larval animal.[2]

The cysticerci seem to occur pretty frequently in both sexes. They may be met with also at all ages beyond infancy, though, as Cruveilhier has remarked, they seem to be most frequent in the latter half of life, and have often been met with in very old people.[3]

b. Hydatids.[4]—In the brain, as in other organs, the hydatid or hydatids are mostly enclosed within an outer sac or cyst. In this organ, however, it is generally very thin, and in some cases it has been stated to have been altogether absent, the hydatid membrane pressing immediately against the compressed brain tissue. When they occur in the lateral ventricles, the enveloping cyst is always absent. In the great majority of cases, only one hydatid cyst is met with, though this may contain two, three, or more hydatids of different sizes; usually, however, a single cyst exists containing a single hydatid. The size of the cyst varies, generally, from that of a marble up to a large orange, though occasionally this limit is much exceeded. Thus, in a case observed by Mr. Headington and reported by Dr. Abercrombie,[1] an immense hydatid cyst was found within the left lateral ventricle, which nearly extended to the circumference of the brain on the same side, and "contained about sixteen ounces of limpid fluid;" and in another case, recorded by Rendtorff, an enormous mass of hydatids weighing two and a half pounds, was found in the same situation, in a girl only eight years of age. The cyst is frequently lodged in the centre of the white matter of one of the hemispheres, and it may increase in size till it occupies almost the whole of one of the lobes—anterior, middle, or posterior, as the case may be. Occasionally it occupies the greater part of two contiguous lobes, and may project towards the circumference, as well as into the lateral ventricle. In both these situations the cyst may be covered only by a thin layer of nerve substance; or it may be uncovered, owing to the brain tissue having disappeared under the influence of the gradually increasing pressure. I have only found one case on record in which an hydatid cyst was lodged in the substance of the cerebellum; in this instance, however, it was large, measuring three inches by two, projecting into the fourth ventricle, and extending transversely across from the right to the left lobe, so as to be covered by a coating of brain matter at each extremity not thicker than a wafer.

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[1] Traité des Entozoaires. Paris, 1860, p. 657.

[2] Thus constituting the third stage of development, when the animal is termed a *strobilus*. See Brit. Med. Journ. 1859, p. 272, where a specimen, apparently in this stage of development, is recorded to have been met with in connexion with the fourth ventricle.

[3] Dr. Cobbold says that more than 100 cases of cysticerci in the brain are on record. References to many may be found in his *Entozoa*, p. 224, and Professor Griesinger (Archiv der Heilkunde, 1862) has analysed the details of between fifty and sixty of these cases.

[4] The following remarks do not refer in any way to hydatids having their seat in or between the membranes of the brain. From various sources I have ascertained the details of thirty cases of hydatids contained within the cerebrum and cerebellum. I have seen references to a few other cases also, of which I have not been able to ascertain the

Although usually only one hydatid cyst is met with in the brain,

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details. For many of the references I have been indebted to Davaine's Traité des Entozoaires, Dr. Cobbold's Entozoa, and Dr. Ogle's paper in the Med.-Chir. Rev. July 1865, p. 206. The references to these 30 cases are:—1. *Martinet*, Lond. Med. Repos. 1824, vol. ii. p. 408.—2. *Bailey*, Lond. Med. Repos. 1826, vol. ii. p. 144.—3. *Morrah*, Med.-Chir. Trans. vol. ii. p. 262.—4. *Hooper*, Morb. Anat. of Human Brain, 1826, pl. xiv. p. 65.—5. *Dalgleish*, Lancet, 1832, p. 168.—6. *Guérard*, Lancet, 1835, p. 45.—7. *Bree*, Lancet, 1837, p. 53.—8. *Sturton*, Lancet, 1840, p. 494.—9. *Berncastle*, Lancet, 1846, p. 635.—10. *Barker*, Trans. of Path. Soc. 1859, vol. x. p. 6.—11. *Baillarger*, Brit. Med. Jour. 1861, p. 286.—12. *Risdon Bennett*, Med. Times, 1862, p. 80.—13. *Ogle*, Brit. and For. Rev. July 1865, p. 207.—14. St. Thomas's Hosp. Mus. No. 101.—15. St. Barthol. Hosp. Mus. No. 60.—16. *Davaine*, Gaz. Méd. de Paris, 1862.—17. *Abercrombie*, Diseases of the Brain, &c., 3rd ed. 1836, p. 447.—18. *Zeder*, Davaine's Traité des Entoz. 1860, p. 644.—19. *Barth*, Bull. Soc. Anat. ann. xxvii. 1852, p. 108.—20. *Calmeil*, Dict. de Méd. art. ENCÉPHALE, t. xi. p. 588, 1835.—21. *Faton*, Bull. Soc. Anat. 1848, p. 344.—22. *Becquerel*, Gaz. Méd. de Paris, 1837, p. 406.—23. *Rendtorff*, Dissert. de Hydat. cap. 10, p. 22, Berlin, 1822; and *Livois*, Rech. sur les Echinoc. p. 100, Thèse, Paris, 1843.—24. *Cazeaux*, Bull. Soc. Anat. ann. viii. 1833, p. 106.—25. *Carrère*, Dict. de Méd. de Chir. et d'Hygiène Vétérin. 1839, t. vi. p. 157, art. TOURNIS.—26. *Tonnelé*, Bull. Soc. Anat. ann. xxvi. 1851, p. 165, case xxxi.—27. *Chomel*, Gaz. des Hôpit. t. x. 1836, p. 619.—28. *Montansey*, Bull. Soc. Anat. ann. ii. 1827, p. 188.—29. *Aran*, Arch. Gén. de Méd. 3me. Sér. t. xii. 1841, p. 98.—30. *Leroux*, Cours sur les Génér. de Méd. prat. t. ii. p. 12, Paris, 1825.

[1] Diseases of the Brain, &c. 3d. edit. 1836, p. 447, case xxxiii.

still sometimes two or three, or even many, are encountered in different parts of the organ. In these cases their size is generally in the inverse proportion to their number; so that in some instances, instead of meeting with one large cyst, such as we have before alluded to, we encounter a number of little ones varying in size between a mustard seed and a hazel-nut.[1]

As an instance of multiple hydatids I may refer to a case recorded by Léveillé, and quoted by Davaine, in which many were found in the meninges and at the surface of the brain, in the corpus callosum, in the left middle cerebral lobe, in the right optic thalamus, and in other parts.

The increase in size of the hydatid being usually slow and gradual, little or no change is generally observed in the surrounding brain substance, which gradually atrophies under the pressure of the growing cyst. But occasionally congestion or softening does occur in the surrounding brain tissue, and, more rarely still, the presence of an hydatid in some portion of the brain seems to excite changes in the whole organ, and even in the cranium. Thus in the case of hydatid in the cerebellum, before alluded to, occurring in a man 24 years of age, the brain was found to be denser and firmer than usual, the ventricles distended with four ounces of clear fluid, and the skull-cap extremely thin, having a medium thickness of not more than about $\frac{1}{10}''$, and at the squamous portions of the temporal bone being quite wafer-like, and not more than $\frac{1}{30}''$ in thickness. In a remarkable case recorded by Dr. Barker (10): "The calvaria was healthy but exceedingly thin, so as to be transparent in numerous places; the outer surface was natural, but the inner presented a series of shallow depressions, separated by angular ridges, evidently produced by the long-continued pressure of the subjacent convolutions, of which they presented an accurate mould. The surface also was congested, rough, and softer than natural. The base of the skull and its dura mater were healthy. There was no sub-arachnoid fluid, the convolutions being compressed against each other, and against the parietes, so as to obliterate the sulci; the surface was not congested. In the posterior lobe of the right cerebral hemisphere was a hydatid cyst, occupying nearly the whole lobe, which was thus converted into a fluctuating cyst. It had rendered the lobe irregular, and lobulated, and increased its dimensions; but the hydatid was covered everywhere by brain substance, although in many situations it was a mere film. The lobulated character seemed to have been produced by the superficial veins acting as ligatures. The cyst was single, about as large as a middling-sized orange, and contained two hydatids, one nearly as large as the cyst itself, the other the size of a walnut. They contained no secondary cysts; the brain in all other respects was healthy."

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[1] In cases of multiple hydatids, their small size may be explained by the fatal nature of the malady, and the early death of the patient. The duration of the life of the patient naturally varies according to the situation and number of the hydatids. Davaine records one case (loc. cit. p. 650) in which a large single cyst must have been four years old.

The hydatids met with in the brain are almost always barren, and thus correspond with the *acephalocysts* of Laennec. Sometimes they are perfectly simple, but they may contain smaller secondary cysts in their interior, or bear them as buds on their exterior surface. The hydatids usually contain a clear, limpid fluid, and their walls are made up of the usual thin, structureless, and concentrically arranged lamellæ. In only two (12 and 23) out of the thirty cases of which I have read the details, is any mention made of the hydatids containing echinococci or their remains. In these fertile cysts, in addition to the echinococci, the remains of the fibro-granular germinal membrane may be detected on the internal surface of the hydatid. Many cases of so-called hydatids in the brain are recorded by old writers, which have but a very doubtful right to this title. The word was formerly used with great laxity: everything in the shape of a cyst receiving this appellation—even the vesicles so common in the choroid plexus, which are now known, in the great majority of cases, to be simple serous cysts.

Hydatids in the brain seem to occur as often in the one sex as in the other. As regards time of life, they seem to be met with, in the great majority of cases, in individuals between the ages of 10 and 30 years. I have found the age of the patient stated in 24 out of 30 cases: of these, 3 were below 10 years of age (5, 7, and 8 years), and 3 above 30 years (one "middle age," 37 and 38), whilst the remaining 18 were between the ages of 10 and 30 inclusive.[1] This is very notable, and in striking contrast with what is known concerning the cysticercus and its tendency to occur rather in the latter half of life, than in younger individuals. We know so little as to the mode in which the human body becomes infected with these cystic entozoa, that it is extremely difficult to explain such peculiarities. We do know, however, that the adult or fully developed condition of the echinococcus hydatid exists in the intestines of the dog, though, of course, not in that of all dogs, and perhaps we may also say that individuals between the ages of 10 and 20 years, have generally more to do with these animals than those of an earlier or more advanced age. This is a mere suggestion which, unfortunately, we are unable further to develop.

19. *Silver and Lead.*—In the 11th vol. of the Transactions of the Pathological Society an account is given by Mr. Sydney Jones of the post-mortem appearances in an old epileptic, who had for several years been in the habit of taking nitrate of silver as a remedy. "The choroid plexuses were remarkably dark: from their surface could be scraped a brownish black soot-like material; a similar substance was found lying quite free in the cavity of the fourth ventricle, apparently detached from the choroid plexus." A specimen of metallic silver was obtained from the plexus.

Lead has several times been detected by the aid of chemical analysis in the brain.

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[1] Several cases of hydatids in the membranes of the brain, of which I have read, have, however, been over this age.

Hypertrophy.—The accounts given by Dance,[1] Andral,[2] and Rokitansky,[3] of the morbid appearance presented in the so-called hypertrophy of the brain, are so harmonious and striking as to point undoubtedly to some definite structural modification, differing altogether from the enlargement due to congestion. A similar condition has also been noticed, and more briefly alluded to, by Bouillaud[4] and Laennec.[5]

Dance and Andral give the post-mortem appearances met with in seven adults, one of whom was 39 years of age, whilst the others varied between 26 and 30. In these examinations the following pathological conditions were met with :—The skull was of average size and shape ; a great turgescence of the brain was noticed on the removal of the skull-cap, which became even more manifest when the dura mater was cut; the dura mater itself was rather thin, and the arachnoid and pia mater remarkably exsanguine, free from all moisture, and easily torn; the convolutions were completely flattened, and separated only by small lines of demarcation, instead of well-marked sulci; on section, the substance of the brain was found to be extremely anæmic, with much less than the usual distinction between the grey and the white matter, owing to the extreme paleness of the former ; the white substance presented an almost completely bloodless section, whilst its density and consistence were so much increased as to make it comparable to "the white of an egg hardened by boiling;" the ventricular cavities were very small, and quite devoid of fluid; and lastly, these changes, though affecting the whole of the cerebrum, did not extend to the cerebellum, pons, medulla, and cord, all of which had their natural consistence.

But there is another form of "hypertrophy" of the brain, which is of much more frequent occurrence. This is met with in some young children, who present obvious marks of being rickety, and is usually indicated by the existence of more or less enlargement and alteration in the shape of the cranium. The skull becomes especially prominent in the frontal region, and often approximates somewhat to the form met with in hydrocephalus. In this variety of hypertrophy, also, there is more or less compression of the brain, as indicated by the existence of anatomical characters similar to, though less strongly marked than those already described.

Whether any relationship exists between the hypertrophy of the brain as it occurs in adults, and that which occurs in childhood, is not known. Nor are we better informed as to the precise nature of the histological change. We neither know whether it is the same in both cases, nor what it is in either. Most pathologists seem to agree in the supposition, that the increased bulk is due to an augmentation of the interstitial substance or neuroglia of the cerebral hemispheres, rather than to an increase in the number or size of the proper nerve elements. So that, if this be true, the disease cannot be looked upon as a hypertrophy of the brain in any strict sense of the term. It has been held by Rokitansky that there is an actual increase in the amount of neuroglia, whilst Sir William Jenner and others are of opinion that, in childhood at least, and when associated with rickets, the enlargement of the brain is due to an infiltration, more particularly of the anterior lobes, with an albumenoid material similar to that met with in the liver, spleen, and other organs. If this be the case, then the brain substance ought to yield the ordinary reaction with iodine; and the characteristic changes in the coats of the arteries, peculiar to this albumenoid degeneration, should be recognisable. Accurate and careful microscopic observations as to the nature of the morbid changes have yet to be made, and without these no real light can be thrown upon the pathology of these remarkable affections.

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[1] Répert. d'Anat. Patholog. par Breschet, 1828.
[2] Clinique Médicale, Trans. by Spillan, 1836, p. 174.
[3] Patholog. Anat. (Syd. Soc.), vol. iii. p. 373.
[4] Traité de l' Encéphalite. Paris, 1825.
[5] Journal de Méd. de Chirurg. et de Pharm. t. xi. p. 669.

There are, however, two other cases of hypertrophy of the brain on record, which differ notably in all respects from the forms to which I have just alluded. In both there was great enlargement of the cranium as well as of the brain; and, owing to this coincident enlargement of the brain and its case, there was not only an absence of the signs of compression of this organ during life, but also an utter absence of the pathological appearances peculiar to the other forms of hypertrophy. The particulars of one case, that of a child who died at the age of five years, have been narrated by Dr. Scoutetten;[1] whilst those of the other, a patient of Dr. Sweetman, who died when a little more than two years of age, have been detailed by Sir Thomas Watson.[2] In both these children, the head equalled that of an adult in size, the skull was somewhat thickened, the dura mater was unduly adherent to it, the arachnoid was moist, the pia mater fully injected, and the convolutions not at all flattened. The ventricles were small: in M. Scoutetten's case they contained a very slight amount of reddish serum, whilst in Dr. Sweetman's they were empty. In this latter case, also, the surfaces "of the medullary matter, exposed by repeated sections, presented very unusual vascularity." Nothing is said concerning the amount of vascularity in M. Scoutetten's case, and in neither of the reports is any mention made of an undue consistence or alteration in density of the nerve matter. The amount of brain substance above and behind the ventricles seemed to be more especially increased in the elder child, since to reach these, from above, an incision nearly three inches in depth was required. Regarding the nature and cause of this enlargement, we know even less than concerning the other forms. Is there an actual increased growth of brain substance—including a due proportion of nerve element proper, and of interstitial substance? or does the increased bulk, in these cases also, result from an augmentation in bulk of the neuroglia alone? and even if this be the case, we may still inquire as to the nature of the change which it has undergone.

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[1] Archiv. Gén. de Méd. 1825, t. vii. p. 44.

[2] Lect. on Princip. and Pract. of Physic. 4th ed., 1857, p. 427.

Much doubt also exists with regard to partial hypertrophies of the brain. Whilst admitting their extreme rarity, Rokitansky says:—"There can be no question that smaller portions of the brain really are separately hypertrophied. Many of the observations brought forward as instances of this occurrence are undoubtedly erroneous; adventitious formations infiltrated through the cerebral tissue may have occasioned at once the enlargement and the error. There are, however, some instances which may be relied on, in which the optic thalamus and the pons were hypertrophied; and I have myself also met with a most remarkable case of hypertrophy of the medulla oblongata." Andral[1] also believed in the existence of a limited local hypertrophy of parts of the brain. These so-called hypertrophies were, in all probability, produced by a hyperplasia of the interstitial tissue of the parts, though it seems more than questionable whether we ought to follow Rokitansky, and apply this name to an increase of bulk which has been thus occasioned.

PROGNOSIS.—In all cases when the presence of an actual growth within the cranium is diagnosticated, the prognosis is very grave, but the degree of gravity depends upon the nature of the growth rather than upon the character or intensity of the special symptoms which it has occasioned. Thus, if there be reason to believe in the existence of carcinoma, the future prospects are as bad as they can be; if the conclusion be that syphilis is the cause of symptoms, there may be room for hope of complete recovery. Without entering upon a discussion of the general grounds of prognosis in the several cachexiæ which have been enumerated (p. 497), as the cause of tumour, it may be well to direct attention to a few points with regard to some of them.

If tubercle is believed to exist, the prognosis is highly unfavourable; but the course of tubercle *en masse* in the brain is sometimes exceedingly slow, and this is the case especially in children. The advance towards a fatal issue is to be apprehended when there is marked hectic, much elevation of temperature, and when the symptoms indicate the progressive invasion of different portions of the brain. On the other hand, when—although there may be distinct paralysis, or amaurosis—the general health is tolerably good, and the symptoms have shown but little tendency to increase in either intensity or extent, there may be considerable prolongation of life.

The hypertrophy of the brain which is met with in children is slowly progressive, but its prognosis, under all rates of advance, is eminently unfavourable.

In syphilitic diseases of the brain or its meninges there is much room for hope; and it seems to be of little moment that the symptoms are varied and severe. Those which are the least amenable to treatment are the losses of sight and hearing, which not unfrequently exist; paralyses and spasmodic affections are often removed with considerable rapidity. The length of time during which the symptoms have lasted is a further guide in the prognosis, the hope of restoration being in inverse proportion to the duration of the morbid state. Still, unless the general condition be one of highly marked cachexia, amendment may be confidently expected. The presence of disease in the kidneys is of unfavourable omen, but even it often disappears under an antisyphilitic treatment. There are no cases which appear so bad and which recover so well as some examples of intracranial syphilis. Until the diagnosis of the constitutional state is established, the case may appear absolutely hopeless; sometimes the only missing link in the history may be unattainable because the patient is insensible, or in such a state of mental incapacity that no reliance can be placed on his assertions, but yet from such condition he may completely recover.

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[1] Précis d'Anat. Patholog. t. iii. 776.

In the case of lead-poisoning the prognosis is favourable, provided that after a few applications of either the continuous current, or of faradisation, the muscles show some remnant of irritability. When the paralysis has existed for a number of years, and the wasting of muscular tissue is very great, it may be impossible to restore the limb, but yet, by continuous treatment, the advance of symptoms may be arrested.

In those cases where there is reason to suspect the existence of either aneurism, hydatids, or carcinoma, the prognosis is eminently unfavourable; but the forecast of a fatal termination is to be based upon the state of the general health of the patient, rather than upon the special cerebral symptoms.

Under all conditions of Adventitious Product uncontrollable pain and vomiting are the most unfavourable symptoms; the former deprives the patient of rest, and the latter renders food useless, and often worse than useless, through the fatigue occasioned by its rejection.

TREATMENT.—There is nothing special which can be said with regard to the treatment of adventitious products in the head, for under all circumstances it is simply that of the different dyscrasiæ upon which they depend. The only remark which it seems to me desirable to make, is one in favour of the administration of large doses of iodide of potassium when there is a belief in the existence of syphilis. I have repeatedly seen the most menacing symptoms removed by the exhibition of K. I. in doses of forty grains, three and four times daily. When this has failed, recourse to mercury has proved curative, and with especial frequency when in conjunction with the baths and waters of Aix-la-Chapelle. Small doses varying from $\frac{1}{24}$ to $\frac{1}{12}$ of a grain of the red iodide of mercury have appeared to me to be more generally useful than any other form of mercury; but when even these doses cannot be borne by the stomach—an event which rarely happens

—happy results may follow the exhibition of mercury by fumigation or inunction.

The pain of cerebral tumour may be palliated, and sometimes removed altogether by Indian hemp, or the application of ice; and sleep may be obtained by the chloral hydrate.

Sickness is sometimes treated most successfully by absolute rest to the stomach, the patient being fed by nutritive enemata.

Convulsions may be checked by bromide of potassium, in doses of ten or twenty grains; but the powers of the therapeutic art are, with the exceptions above mentioned, enclosed within painfully narrow limits, and all that can be done is to palliate evils which cannot be removed.

CEREBRAL HÆMORRHAGE AND APOPLEXY.

By J. Hughlings Jackson, M.D., F.R.C.P.

The text[1] of this article is Cerebral Hæmorrhage, using the term in the sense of escape of blood within the intracranial nervous centres. Since very little or very much blood may be effused, the symptoms vary extremely in degree; and since the parts in which rupture of vessels may take place are numerous, the symptoms vary much in kind. When large and rapid effusion occurs, there is the apoplectic condition. Cerebral Hæmorrhage is one cause, and the most frequent cause of apoplexy. So frequently does hæmorrhage cause apoplexy, that this name has got into use for hæmorrhages in other organs. Thus we speak of Retinal Apoplexy and of Pulmonary Apoplexy. Since this use or abuse of the word leads to confusion, the term is often qualified by the word Cerebral. We shall restrict Cerebral Hæmorrhage to effusion of blood into the brain, and reserve the word Apoplexy for the comatose condition which large effusion of blood and other causes produce. Cerebral Hæmorrhage will be chiefly considered, and other causes of Apoplexy will be spoken of under the head of diagnosis. It will be absolutely necessary to make occasional reference to meningeal hæmorrhage, although this has been considered in another part of this volume, since meningeal as well as Cerebral Hæmorrhage may produce Apoplexy.

Morbid Anatomy.

Position.—The effusion of blood is circumscribed. With rare exceptions it occurs in but one side of the brain, nearly always in a limited part of that side, most frequently in the ganglia at the base. Occasionally, however, it breaks out of the substance of the brain, usually into the lateral ventricle, but sometimes on to the surface. It is of little use to take as a basis for statistics, records of published cases; from the Pathological Society's Transactions, for instance. No one would think of bringing before the Pathological Society an ordinary specimen of Cerebral Hæmorrhage. Cases of special interest are published, such for instance as cases of hæmorrhage into the pons Varolii simulating opium poisoning. A goodly number of cases of large hæmorrhage into the cerebellum could be collected from medical periodicals, as many such cases are pretty sure to get into print. But large hæmorrhage into the cerebellum is in reality exceedingly rare. During the seven years I have been attached to the London Hospital, I have seen but two cases. Again, statistics take count mostly of *fatal* cases of hæmorrhage—of effusions of blood into the brain big enough to kill quickly—and of comparatively few of those cases so frequently seen in hospitals of hemiplegic patients who have recovered from coma, and who go out of the hospital with more or less permanent palsy. Many of these patients die months or years after in workhouses. If we speak of hospital cases only, we thus exclude many cases of very great clinical importance, and besides those mentioned we exclude cases of rapid death from meningeal hæmorrhage, for most of such cases are seen by those engaged in private practice. For these reasons I do not give nor refer to statistics, but it will, I think, be safe to say that *large* hæmorrhage very often occurs in the corpus striatum and thalamus, often in the pons, rarely in the cerebrum, very rarely indeed in the cerebellum, and scarcely ever in the spinal cord.

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[1] In the collection of materials towards writing this paper, I have to acknowledge with my warmest thanks the help I have received from Dr. Anthony Roberts, Mr. F. M. Corner, Mr. Steggal, Dr. James Jackson, Mr. George Mackenzie, Mr. Frederick Mackenzie, Mr. Grubb, Mr. Norton, Mr. Llewellyn, Mr. Gordon Brown, Mr. Stephen Mackenzie, Mr. G. E. Herman, Mr. Louis Mackenzie, and others. I have to acknowledge also most valuable assistance in the investigation of cases of apoplexy from my colleagues Dr. Sutton, Dr. Woodman, Mr. Hutchinson, Mr. Waren Tay, and Mr. MacCarthy. I have to thank my other colleagues for their generosity in placing cases at my disposal for investigation. For valuable help in the revisal of this article, I am indebted to my friends Dr. Woodman, Dr. Gowers, and Mr. Stephen Mackenzie.

We occasionally find blood in the lateral ventricles. In the vast majority of cases, it comes from a rent in the corpus striatum or thalamus opticus. It may extend to the fourth ventricle, and escape sometimes on the surface of the brain. I have, however, twice known blood effused into the ventricle without injury of the ganglia in its floor. These were exceptional cases; the blood came from bursting of large aneurisms. In one the aneurism was seated in the middle line of the hinder part of the circle of Willis at the divergence of the posterior cerebral arteries. In the other case an aneurism of a small artery of the posterior lobe had burst into the posterior cornu—blood had escaped also on to the surface of the brain. In the first edition of the article Convulsion, a case occurring in Mr. Gayton's practice was mentioned, the notes of which were supplied by Dr. Woodman. In this case Dr. Woodman found no aneurism, and no laceration of the brain.[1]

Multiple Effusions.—Sometimes two or more recent clots, even large clots, are found in different parts of the brain. My friend Mr. Llewellyn showed me a specimen in which there was a clot in the floor of the lateral ventricle, and another in the pons. The two clots came on at the same time, for the patient had gone to bed as well as usual and died next day of Apoplexy, which began in the night. Dr. John W. Ogle (Pathological Soc. Transactions, vol. xv. p. 8) has recorded a case in which there were three recent clots, one in the right corpus striatum, one in the left thalamus opticus, and one in the pons Varolii. Dr. Baümler has supplied me with notes of the case of a man, thirty-four years of age, who died three hours after a fit, in whose brain four recent clots were found: a large one in the centre of the right hemisphere, a small one in each of the optic thalami, and a small one in the right crus cerebri. The ventricle was also full of blood. After discovering the main clot, we should carefully search the rest of the brain, especially the pons and medulla, for small effusions, often only little specks.

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[1] A man, 24 years of age, died rather suddenly twelve hours after a fit of convulsion. Although this man had recovered so as to take broth and to answer questions, his cerebral ventricles were found at the autopsy full of blood. Mr. Prescott Hewett says that a very, very slight laceration of the floor of the lateral ventricle may, if it correspond to the situation of a large vein, give rise to an extensive extravasation of blood into this cavity (Holmes' Surgery, p. 314). Blood in the fourth ventricle may come from a rent in the pons.

So far we have spoken of recent clots. It is not at all rare to find effusions of blood of very different dates. Sometimes after discovering a recent clot on one side of the brain we may find much of the opposite motor tract damaged by old effusion when there has been no corresponding palsy for some time before death.

Size of Clots.—The size of a clot varies from that of a pea or less, to a mass the size of one's fist or more. Its size will depend of course on the size of the vessel ruptured, and its shape depends somewhat upon this also. In small capillary hæmorrhages the blood may lie in streaks in the brain substance, rather pressing the tissues apart than destroying them. (We see by the ophthalmoscope in many cases of small hæmorrhages into the retinæ, in cases of Bright's disease, that the blood is arranged in the direction of the retinal nerve fibres.) It is doubtful whether capillary hæmorrhages in the brain give rise to symptoms. (A patient's sight may be quite good when there is considerable streaking of his retinæ with blood.) When from a large effusion the fibres are torn, the clot is rounder and more distinct, and gives rise to symptoms.

The larger the clot, not only the greater the local destruction of nerve tissue, but also the more squeezing there is of the parts not directly damaged. When a large quantity of blood has been effused in one side of the brain, we see *post mortem* that the affected cerebral hemisphere looks more voluminous, that the convolutions are flattened, and we find on section that the cerebral substance is more anæmic than on the healthy side. If after sawing round the skull we insert the knife in the line of the saw-cut, and remove the skull cap with the part of the brain it contains—a plan Mr. Hutchinson adopts to display the position, and effects of traumatic hæmorrhage—we may find that the falx bulges to the sound side, and this is evidence that the other hemisphere has been compressed. These facts are of importance with regard to the causation of loss of consciousness from Cerebral Hæmorrhage. (See p. 544.)

Changes in Effused Blood.—We find *post mortem* most varied appearances according to the age of the clot. Soon after its effusion there is seen a soft black jelly mixed at its edges with small specks of brain and lying in a bed of softened brain. In the later stages the appearances are widely different: there is a cyst—"apoplectic cyst"—filled with ochre-yellow fluid, or there is even a cicatrix. We sometimes find the two extremes, a recent black soft clot, and one or more cysts the relics of old effusions. In the progress from the recent clot to the apoplectic cyst, we have to consider changes in the blood effused and changes in the brain about it.

On removing as much as we can of a recent clot we mostly see, especially when the effusion is in the corpus striatum and thalamus, an irregularly-shaped cavity with a shaggy wall of soft brain intermixed for a short distance with specks of blood. The local softening results partly from imbibition of serum from the clot and partly from inflammatory changes excited by the clot. The inflammation—local encephalitis—may lead to extensive disintegration of brain ("the apoplectic clot is even transformed to an abscess of the brain," Niemeyer), but usually the process is limited; it is conservative, and leads to the formation of the cyst wall to be presently mentioned.

The progress to the final stage is gradual. The clot diminishes in bulk, becomes softer and browner; next the colour becomes yellow; granules of blood pigment and hæmatoidin crystals form. Finally the clot is represented by thin ochre-yellow, or even clear, fluid. Simultaneously, as a result of a slow inflammatory change, the wall of the cavity undergoes great alteration. An organized membrane forms from the neuroglia, and the apoplectic *cyst* results. This is the most common termination, but the process may go even further. There may be no cyst, but a hard pigmented patch, an "apoplectic cicatrix."

Traumatic hæmorrhage.—In cases of injury the clot mostly affects the surface, and most frequently the convolutions of the base; there is rather a pulp of brain and blood than a distinct clot. An injury is to be suspected whenever blood is found effused close to the convolutions, especially if these be bruised, and if there are many small specks of black blood near the principal clot; above all, if distant from the principal lesion there are very many little specks of black blood intermixed with, and round about pulpy patches.

At post-mortem examinations, especially of those who have died of chronic Bright's disease, we occasionally find evidence of former hæmorrhage into the grey matter of the convolutions. There is often a cup-like depression with hardened walls containing ochre-yellow fluid. But if these be in several parts which we know to be often bruised by injuries to the head,—for instance, on the under surface of the anterior or sphenoidal lobes,—it is probable that there has been injury to the head. (See Bristowe, Path. Soc. Trans. 1869-70.)

Rupture of Aneurisms of large Cerebral Arteries.—We have to speak of aneurisms of the small arteries of the brain (miliary aneurisms, p. 507), but occasionally Cerebral Hæmorrhage results from rupture of aneurism of the large arteries at the base. As a rule, however, their rupture produces meningeal hæmorrhage. (See arts. Adventitious Products, Meningeal Hæmorrhage, and Convulsions.)

Hæmorrhage from Cerebral Tumours.—Cerebral Hæmorrhage has occasionally, but very rarely, its origin from vascular tumours of the brain. I have recorded three such cases in the *Lancet*, Oct. 29, 1869. The tumour is the glioma of Virchow. As he points out, ordinary Cerebral Hæmorrhage has its seats of election, and these are not precisely the places where glioma is most frequently found. Glioma occurs most frequently in the white mass of the hemisphere, especially in the posterior and anterior lobes, places where ordinary Cerebral Hæmorrhage is rare. It is very important to bear in mind a remark he makes to the effect that it often requires a very attentive examination to distinguish hæmorrhagic gliomata from traumatic hæmorrhage, from red softening, and from rupture of cerebral aneurism. (See art. Adventitious Products and art. Softening.)

Lungs.—Of course all organs are to be examined *post mortem* for, as we shall see, the heart and kidneys are often diseased. There are, however, often striking post-mortem appearances in the lungs, which are owing to the apoplectic condition. These are not peculiar to apoplexy from Cerebral Hæmorrhage, and, indeed, some of them at least occur in other modes of dying. They are very varied, and the variation depends on two factors—the rapidity of death and the condition of the patient (his age and state of health) when taken ill. I have not been able to make out any difference from *position* of the Cerebral Hæmorrhage. I have known the lungs pale like "cholera lungs," and weighing only twenty-two ounces, in a patient who died in an hour of large Cerebral Hæmorrhage. But as a rule even in patients who die more quickly—as in some cases of meningeal hæmorrhage—the lungs are congested. They are often bulky and œdematous in their dependent parts, which easily break under pressure, and very emphysematous (vicariously) in front. If the patient be a robust, full-blooded man, the lungs may be black, from cramming with blood up to their anterior margins, and easily breaking into a pulp, as in cases of rapid death from traumatic hæmorrhage. In some cases of slower death, we find pulmonary apoplexies. I have in two cases (one traumatic and one opium-poisoning; both patients young) seen lungs which on section looked like the "damson lungs" of heart disease. In other cases where patients die very slowly, we find on section granite-coloured patches of various sizes slightly raised and well-margined. These lobular patches are often called lobular pneumonia, and when very numerous and almost confluent, the word pneumonia is sometimes used without any qualification. Yet these changes occur with rare exceptions, in both lungs and in the dependent parts of all lobes, and ought not to take the name which belongs to a well-marked independent disease. They may affect one lung more than the other,[1]—

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[1] In one case, that of a woman aged 73, who died in forty-seven hours, of hæmorrhage into the substance of the left cerebral lobe; one lung, which was universally adherent,

the right usually. Since, when we find these granite-coloured patches, we occasionally find recent apoplexies also, and indeed patches of intermediate colour, I believe they are, as Brown-Séquard has stated, altered apoplexies. Sometimes one or more of them are broken down into a grumous pulp; over some nodules near the surface there may be slight pleurisy.

ETIOLOGY AND PATHOLOGY.

In speaking of Etiology and Pathology we for the present exclude cases where the bleeding is the result of injury to the head, where it occurs from rupture of an aneurism of a *large* cerebral artery, such as the middle cerebral or basilar, or where it starts from a vascular tumour of the brain. We cannot speak at all of cases of intracranial hæmorrhage occurring in purpura (see art. Purpura, Vol. I. p. 793), or scorbutus (see art. Scorbutus, Vol. I. p. 784), or pyæmia (see art. Pyæmia, Vol. I. p. 561), nor of "red softening" (art. Softening, Vol. II.). Obviously the above are in their etiology and pathology very different things; they only agree in that there is escape of blood in or upon the brain. Moreover they differ much clinically. We consider, in what follows, the common run of cases.

Age.—Cerebral Hæmorrhage rarely occurs in persons under forty. This age is that at which one of the most important factors in the causation of Cerebral Hæmorrhage, degeneration of arteries, begins to be common. Changes in the arteries of old men are scarcely to be considered morbid. "To degenerate and die is as normal as to be developed and live" (Paget). In the progress to healthy old age the body, *as a whole*, descends in vitality; the blood wastes, numerous capillaries obliterate, the lymphatic system undergoes involution, there is senile emphysema. Although the heart becomes somewhat bigger as years increase, there is not excessive cardiac hypertrophy. There is an increase in bulk of the whole organ, not an extreme hypertrophy of the left ventricle only, as there so often is in persons of middle age who die of Cerebral Hæmorrhage. The degenerations we are especially concerned with in this article are premature, moreover they are often attended by disease of the kidneys. In a patient under forty the arteries may feel tougher than the arteries of another person of eighty.

Some of the exceptional cases of large Cerebral Hæmorrhage in *young* people whose arterial system, as a whole, has not undergone degenerative changes, are cases of rupture of aneurisms of the larger cerebral vessels; for instance, of a branch of the middle cerebral or of the basilar. Indeed, if there be no evidence of the arterial and other degenerations in the body, to be presently mentioned, the probability

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was solid by this sort of change; the other, which was not adherent, except by one or two tags, was a good specimen of senile emphysema, and presented scarcely any other morbid appearance.

is that large Cerebral Hæmorrhage in young people (excluding cases of injury, purpura, and the like) is thus caused. Apart from such quasi-accidental cases, we must observe further that the matter of importance in considering the influence of age in diagnosis is to note the general constitutional state the patient has arrived at rather than the number of years he has lived. For Cerebral Hæmorrhage quite like that which occurs so often after forty does sometimes occur at the age of twenty and even under, in people who are subjects of chronic Bright's disease, who have degenerated arteries and hypertrophy of the left ventricle of the heart. I have recorded such a case (London Hospital Reports, vol. iv. p. 337).

Heredity.—It is asserted that in some families there is a tendency to Cerebral Hæmorrhage. Obviously the transmission cannot be of a tendency to certain symptoms—hemiplegia and Apoplexy—but to certain tissue degenerations most strikingly manifested in the arteries. Cerebral Hæmorrhage therefore can be inherited only indirectly. Much used to be said of a certain inherited build of body and of the "apoplectic constitution." Little importance is now-a-days attached to this. Austin Flint says, "The larger number of persons attacked are either spare or of an ordinary build." Niemeyer says, "There is no such thing as an apoplectic constitution indicated by a short neck and broad shoulders."

CONSTITUTIONAL STATE PRIOR TO CEREBRAL HÆMORRHAGE.—It is convenient to consider an extreme case in order that we may state the whole of the factors which *may be* concerned in causing Cerebral Hæmorrhage. We are, as was said at page 526, not now considering cases of hæmorrhage from rupture of aneurisms of the large cerebral arteries, from injuries, or from the like quasi-accidental causes.

At autopsies on patients who have died of Cerebral Hæmorrhage we frequently, if not mostly, find three things: hypertrophy of the heart, chiefly the left ventricle, chronic renal disease, and degenerated[1] arteries (Bright, Johnson, Kirkes). A patient so much and so widely diseased has not a liability to Cerebral Hæmorrhage only; he is liable to inflammation of serous membranes, to bronchial catarrh, to œdema of the lungs, &c. But in this article we have only to do with the triple association as it bears on Cerebral Hæmorrhage. We are especially concerned with the condition of the vascular system, and can only speak incidentally of the renal disease. We have two tasks. Going the natural round of the circulation (heart, large arteries, small arteries, capillaries (and nervous tissue), venous system and lungs), we have first to consider the abnormal conditions of different parts of the vascular system, and next how, from the sums of these several conditions, it results that rupture of the smaller cerebral arteries is determined.

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[1] Retinal changes will be alluded to (p. 550). The careful clinical observer considers minor degenerative changes,—baldness, greyness, state of skin, worn teeth. He inquires for history of gout, and of intemperance.

Heart.—The hypertrophy is of the left ventricle; it is of the kind called simple, because, although the wall of the ventricle is thickened, there is not dilatation; the capacity of the ventricle is, at all events, but slightly increased. Yet, as in other kinds of cardiac hypertrophy, dilatation may ensue. In patients long bedridden from palsy due to Cerebral Hæmorrhage, we may find the heart decreased in size, notwithstanding that there is chronic renal disease.

In this form of hypertrophy there is obviously increased power acting on the *arterial* side of the circulation, and consequently we infer obstruction somewhere in the arterial system. We limit ourselves to cases where there is no obstruction at the aortic orifice, and no incompetence of the aortic valves.[1] The resistance to the heart is much further on. It is peripheral. Although this is agreed on, there are great differences of opinion as to the exact nature of the peripheral obstruction. Dr. Bright suggested that the "altered quality of the blood might so affect the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system." He suggested also that the blood in Bright's disease might act as an unwonted stimulus to the heart. Dr. George Johnson, believing that the blood in renal disease is more or less noxious to the tissues since it contains "urinary excreta," considers that its passage into the capillaries is resisted by *contraction of the small arteries*—the vessels most rich in muscular tissue. The muscular coats of these vessels therefore are hypertrophied in *antagonism* to the heart. Since the small arteries are hypertrophied throughout the body, the obstructions, though each is slight, are in their sum total so large, that in order that the circulation may be carried on efficiently, hypertrophy of the heart must ensue.

But whilst Dr. Johnson believes that the thickening of the walls of the small arteries is genuine hypertrophy, "an increased growth of a normal tissue without change of texture," Dr. Beale doubts whether there is real hypertrophy of the muscular fibre cells, and supposes that the changes in the small arteries are degenerative. He remarks[2] that "there is an increased bulk with altered structure, not simply increased bulk without change of structure (hypertrophy)."

Traube considers that the cardiac hypertrophy in Bright's disease is a consequence of increased tension of the arterial circulation, partly the result of the diminished calibre of the renal circulation, and partly the result of greater volume of the blood from diminished excretion of fluid. The objection which has been raised to this view is, that the hypertrophy of the heart mostly begins in the earlier stages of Bright's disease.

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[1] For intracranial hæmorrhage is not common in cases of *valvular* disease of the heart, excluding cases of aneurisms of the larger cerebral arteries, and ruptures of these usually produce meningeal hæmorrhage. My observations confirm the statement of Dr. Austin Flint, that important nervous symptoms of any kind—excluding, of course, those produced by the process of embolism—are not common in cases of valvular diseases of the heart (Diseases of the Heart, 2nd ed., p. 180).

[2] Beale on the Urine, 3rd ed., p. 72.

Occasionally, although rarely, we find great hypertrophy of the left ventricle in cases of Cerebral Hæmorrhage, when there is neither obstruction at the aortic orifice, nor renal disease, but simply wide-spread degeneration of the arteries. From these cases it seems clear that degeneration of vessels is a sufficient cause of hypertrophy of the left ventricle. It must be admitted that there are cases of hypertrophy of the heart, which we are unable to explain. We do mostly, however, find hypertrophy of the left ventricle in cases of Cerebral Hæmorrhage.

Large Arteries.—From a degenerative[1] change the large arteries lose much of their chief property—elasticity; they become permanently wider, longer, and more tortuous. We see them move in curves on the temples, and we feel that they are tough and sometimes even "bony." When the large arteries which we can see and feel are thus changed, it is a reasonable inference, that the large arteries of the brain are similarly, although not perhaps equally, altered. Elasticity of the arteries, although it adds no new force, is an important aid to the circulation in equalizing the flow of the blood, gradually reducing it from intermittence in the large arteries to a nearly continuous stream in the smaller arteries. We readily understand, therefore, that the *absence* of elasticity of the larger arteries will be an important factor in leading to rupture of the smaller arteries. The circulation is carried on too much in systole. The smaller arteries will receive the impulse from the strong left ventricle intermittently, not remittently.

Small Arteries.—Here rupture mostly takes place, always in large hæmorrhages. We have to consider several pathological conditions of the smaller arteries. (*a*) Fatty degeneration. Arteries of small size have a highly developed muscular coat, and this coat especially is the seat of fatty degeneration, a change which we may suppose will allow rupture when the vessels are unduly strained. However, not so much importance is attached to this pathological condition as was wont to be, for fatty degeneration is found sometimes in the arteries of very young people. Indeed, Billroth and Bouchard consider it to be most frequently a *result* rather than a cause of cerebral lesions. But even if so, degeneration of arteries, for instance, in a focus as softening, may favour the occurrence of hæmorrhage into the part diseased. (*b*) Charcot, Bouchard, and Charlton Bastian have described what they term "miliary aneurisms;"[1] aneurisms mostly visible to the naked eye, of the size of a pin's head more or less. (These have been fully described, art. Adventitious Products.)

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[1] I speak of *results* under the general term "degeneration." Virchow (Cellular Pathology, Dr. Chance's translation) points out that the true atheromatous change in arteries begins by a slow inflammatory change of the tunica intima—an endarteritis strictly analogous to the active inflammatory changes in endocarditis. I have in this article, however, to do with changes in the vessels so far only as, by affecting the dynamics of the circulation, they favour cerebral hæmorrhage. I have not to do with the processes by which these changes are arrived at; therefore I use the general term "degenerative," which, with the above qualifications, need not mislead. I conclude the footnote by a quotation from an able lecture by Moxon, Med. Times and Gazette, Nov. 12, 1870:—"It is too much the fashion, at least in this country, to assume that all the processes in the arteries which lead to the deformation of their interior by yellow patches, swellings, petrifactions, or erosions, or to aneurisms or rupture of the vessels, are all of a degenerative origin, and that all are sufficiently described and defined in the common notions of atheroma. The truth is, that sub-inflammatory irritation plays a very important part in these changes." Since this was written, Moxon has considered the whole subject in a valuable article in Guy's Hospital Reports, 1870-71.

Capillaries.—These vessels may be found the seats of fatty degeneration. But ruptures allowing large hæmorrhages do not occur here. If it be degeneration of the smaller arteries which produces obstruction and thus induces hypertrophy of the left ventricle of the heart, the diseased state of the smaller arteries may protect the capillaries from strain.[2]

Nervous Tissues.—The influence which changes in the tissues outside the vessels may exert in the causation of Cerebral Hæmorrhage may be most conveniently considered here.

When the brain wastes slowly, there is, in order that the cranium may continue full, compensation. There is, in some cases of wasting of the brain, thickening, genuine hypertrophy, of the skull (Paget). There is, sometimes, increase backward of the capacity of the frontal sinus (Holden); but the most important compensation is by increase in the quantity of the cerebro-spinal fluid. To these sources of compensation Leubuscher and Niemeyer add dilatation of the vessels which will, they believe, favour rupture. And Niemeyer suggests that the frequency of Cerebral Hæmorrhage, in advanced life, depends at least partly on the dilatation of the vessels induced by atrophy of the brain. Further, he thinks that since Cerebral Hæmorrhage leads secondarily to atrophy of the brain, one attack favours the occurrence of another attack.

Softening of the brain, by diminishing the support of vessels, may favour hæmorrhage, but it is almost universally agreed on that the softening we find *post mortem* near to a clot is nearly always the result of the effusion, not a pathological condition prior to the occurrence of hæmorrhage.

Venous System.—We shall speak only of obstruction to the return of the blood from the brain by changes in the lungs, excluding cases like phthisis and senile emphysema, in which the volume of the blood is reduced, and in which, therefore, the pulmonary impediment is to a great extent compensated. From hypertrophous emphysema there results universal peripheral congestion, consequent on the difficulty the venous blood encounters in passing through the lungs—the vascular area of which is reduced by obliteration of a great number of capillaries.[1] From theoretical considerations therefore we might at first glance attach much importance to emphysema as a factor in the causation of Cerebral Hæmorrhage. "In no other disease does the cyanosis attain such severity excepting in cases of disorder of the orifices of the right heart." (Niemeyer.) However, emphysema probably is not often an important factor in the causation of Cerebral Hæmorrhage. There will, in chronic cases, be much compensation by increased power of the right ventricle. Even if there be not full compensation, the left ventricle will have less blood to send to the brain, and thus the *arterial* tension will be diminished. It is the venous system which is overblooded and strained in emphysema; the arterial, from which rupture in Cerebral Hæmorrhage takes place, is underblooded and less strained. In the last stages of emphysema we have, Niemeyer says, the severest symptoms of hyperæmia of the brain. Yet Cerebral Hæmorrhage is a rare termination of this or of any other form of thoracic disease. It is the tension on the venous side of the circulation which is increased in emphysema.[2]

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[1] Dr. Bristowe has drawn attention (Path. Soc. Trans. 1859) to ruptures of *small* aneurisms in the substance of nervous organs as a cause of cerebral hæmorrhage, and gives a drawing of an unruptured aneurism, the size of a grain of wheat, which lay in the substance of the cerebellum. Dr. Henri Lionville has found miliary aneurisms in the retina (Gaz. des Hôpitaux, 1870). See also Dr. Gull (Guy's Hosp. Reports, third series, vol. v. 1859).

[2] Here reference may again be made to Dr. George Johnson's view, mentioned p. 528. In one case of large Cerebral Hæmorrhage he failed to discover any hypertrophy of the small arteries of the brain, but he found them much hypertrophied in the subcutaneous tissue. Inferentially, they were hypertrophied in other organs, although, unfortunately, examinations were made of the arteries of the brain and of the subcutaneous tissue only. In such a case, he suggests that the hæmorrhage results, not from rupture of the arteries, but from rupture of the capillaries of the brain. They rupture because they are not protected from the force of the hypertrophied left ventricle by the hypertrophy of the muscular coat of the arteries delivering blood to them. It is, however, unlikely that rupture of capillaries would cause a large effusion of blood. "The apoplectic fit does not occur in capillary hæmorrhages." (Niemeyer, Text-book of Practical Medicine; translation of the eighth German edition by Drs. Humphreys and Hackley, vol. ii. p. 198.)

The veins are of lower functional structure than the arteries—very few of the cerebral veins contain any muscular tissue—and thus probably they are less liable to disease. The degree of hyperæmia of the face and lips is no exact measure of the degree of venous congestion of the brain in sudden obstruction at least. The cerebral veins are protected from sudden backward strain by the large cerebral sinuses. Nevertheless obstruction at the lungs, especially when occurring quickly, will be a factor, if not an important one, in favouring rupture of cerebral arteries.

We have now to speak of the dynamics of such a person's circulation, in order to show how abnormal strains lead to rupture. To obtain clear ideas upon this point, it is well to run over the peculiarities of the cerebral circulation in health. The brain receives a large supply of blood (a large quantity passes through it), but there are provisions by which it is protected from suddenly increased afflux. There are turns of the carotid and vertebral arteries, the free anastomosis of the circle of Willis, the numerous subdivisions of the arteries beyond that circle, on the convolutions at least, and their small size before they enter the brain substance itself. Perhaps we may add the possibility of diverticular enlargement of the thyroid body, and of the facial arteries, by which in suddenly increased action of the heart the flow to the brain will not be in proportion to the increased quantity of blood sent into the carotid and vertebrals. Further, when there is increased afflux of blood, compensation can occur by diminution of the quantity of the intracranial part of the cerebro-spinal fluid. Some parts of the brain, however, are less protected than others. The arteries which supply the corpus striatum are not capillary in size. They, or many of them, come off from the middle cerebral, which artery is almost the continuation of the internal carotid. Thus its branches lie more in the way of strain from the heart. But on the other hand we have to observe that here there is special compensation. As Hilton points out, the corpus striatum and thalamus (which parts we may presume to have greater and more frequent functional activity than most divisions of the nervous system) lie in great part in the large waterbed of the brain. By this means rapid compensation by diminution of the fluid in the lateral ventricle may occur when these highly vascular parts during their functional exercise swell by becoming for a while more vascular. There is even more. Dr. Bastian (on Tubercular Meningitis, *Edin. Med. Journal*, April 1867), speaking of perivascular canals, suggests (but only as one hypothesis as to their use) that they may constitute " an apparatus for the distribution of cerebro-spinal fluid throughout the structure of the brain, in order that the same protective influence may be exercised over each individual portion of its structure which is exercised over the whole region by that portion of the fluid situated in the subarachnoid spaces." He says too, speaking of perivascular canals, three times the diameter of the vessels they contain,—" This large size of the perivascular sheaths occurs more frequently in the corpus striatum and optic thalamus." Then on the venous side there is the remarkable arrangement of the sinuses, which is such that a backward strain, if it be sudden, will not reach the veins of the brain—or will reach them in a diminished degree. The blood will pass, by preference, so to speak, into the outer parts of the head, face, &c. If the backward pressure be slowly exerted as in chronic emphysema, there will be cerebral hyperæmia, but it will be very slowly developed and very evenly distributed.

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[1] We do not here refer to the acute emphysema which we so often find *post mortem*, and which is especially well-marked in young, robust men who die in the apoplectic condition soon after traumatic intracranial hæmorrhage. In these cases we find great posterior congestion—occasionally pulmonary apoplexies also—and vicarious emphysema of the front parts of the lungs.

[2] Dr. Hyde Salter never saw or heard of Cerebral Hæmorrhage during an attack of asthma or as a result of asthma; for, although here there is acute obstruction to the return of venous blood, the arterial tension is very small: in severe attacks the pulse can scarcely be felt.

Let us suppose that all the abnormal conditions enumerated p. 528 to p. 531 are present. Of course the whole of these factors are not present in all cases. There may be no renal disease, but there are usually degenerated arteries and hypertrophy of the left ventricle. But as the following is chiefly recapitulatory, we shall in its proper order mention each of the conditions which different observers have supposed as well as have proved to be factors in the causation of

Cerebral Hæmorrhage. The small artery is usually the seat of rupture. We take this as the fixed point, and first consider the development of undue strain upon it. Next we speak of negative circumstances, which add to the influence of this strain—of impediments to the exit of blood from the arteries into the veins.

There is a strong left ventricle. The larger arteries are inelastic, and thus the wave of the blood sent forcibly into them is not equalized: as a consequence the impulse from the heart's jerks will be carried on strongly to the smaller arteries of the brain. The smaller arteries of the brain—normally thinner than arteries of other parts—are degenerated. Though this degeneration leads to resistance, it is not the resistance of power; it is a "weak obstinacy." The resisting arteries are fragile and may be actually aneurismal. Moreover if the brain be wasted, the arteries are dilated and less supported. So far for the forward strains.

If we hold that changes of nutrition in the penetralia of the body—in capillary regions—contribute largely to the forces of the circulation (*vis à fronte*), there will be from the imperfect nutrition which the disease of the arteries and capillaries causes (and which in some cases atrophy of the brain signifies), an obstruction (or more precisely a cessation of help) to the flow of blood. The capillaries will not readily empty into the veins. At this point too, as there is often renal disease, we have to recognise, if we follow Bright, Johnson, and Kirkes, a still further element of obstruction in the unwillingness of the tissues to pass impure arterial blood. Further, if there be emphysema or other obstruction at the lungs, the free return of blood from the brain is hindered. Perhaps a condition of plethora, or at least of transient plethora after large eating and drinking, may add to the tension of the circulation by increasing the volume of the blood. If all the above-named conditions be present, there is no wonder that the diseased small arteries, unable to empty readily into the capillaries and veins on the one hand, and jerked by a strong ventricle on the other, sometimes give way. Moreover, when we consider the local peculiarities of the circulation of the corpus striatum and thalamus (p. 532), we can well understand that these bodies are the "seats of election" for large Cerebral Hæmorrhage, notwithstanding the special provisions mentioned p. 532.

The degenerations have been slowly going on, and the diseased vessels are being subjected to increasing strain. A time comes when a vessel more diseased than another, perhaps one the seat of a miliary aneurism, or some vessel specially in the way of strain, mostly a branch to the corpus striatum, gives way. It is not at all rare to hear it said that hæmorrhage is sometimes the result of *obstruction* to the *return* of venous blood from the head in paroxysms of convulsion. Dr. Todd (Diseases of the Nervous System, Lects. vii. and xii.) held this view. But since the hæmorrhage is nearly always in the arterial regions of the brain, and since in the most severe paroxysms of chronic epilepsy, Cerebral Hæmorrhage is excessively rare, it is far more reasonable to suppose that the irruption of blood itself causes the convulsion and the subsequent apoplectic condition. No single instance of actual Cerebral Hæmorrhage in an epileptic fit has presented itself in Reynolds' experience (On Epilepsy, p. 225).

Hæmorrhage from Aneurism of the larger Cerebral Vessels. —In cases of aneurism of the larger cerebral arteries, a very local disease of the vessel may be the sole flaw in the system, excepting perhaps vegetations on the heart's valves (Dr. John W. Ogle and Dr. Church). If there be no misfitting of the valves, no hypertrophy of the heart will ensue, nor indeed any derangement of the dynamics of the circulation of the brain, possibly not even any change in the nutrition of the part supplied by the aneurismal artery if it remains pervious. In a few cases the fatal attack begins, if not in good health, at least in what appears very like good health. The patients feel well and are about at their work. We can say nothing as to their constitutional condition. I have only to do with rupture of these aneurisms in diagnosis. For rupture of them mostly produces meningeal, not cerebral hæmorrhage. (See art. Adventitious Products.)

Localization of Lesions.

We have now to speak of lesions of the motor and sensory tract within the cranium, and of lesions of the two large masses therewith connected—the cerebrum and cerebellum. In this section we consider almost solely paralytic symptoms—those which localize; other non-localizing symptoms will be considered under the head of the Apoplectic Condition (p. 544).

The parts of the motor and sensory tract from above downwards are corpus striatum, thalamus opticus, crus cerebri, pons Varolii, and medulla oblongata. All these parts are double, right and left, although the halves of the last two, pons and medulla, are welded together. In the vast majority of cases, the lesion is of but one *lateral* half of the sensori-motor tract, and its results are one-sided palsy—hemiplegia. In the parts we have spoken of as being welded together, the pons and medulla, the lesion occasionally affects both halves, and thus paralyses both sides of the body. We have to do especially with lesions of one lateral half of the motor and sensory tract at different levels, and consequently with several varieties of hemiplegia. In all these varieties we are concerned with palsies of the limbs, and with palsies of parts supplied by cranial nerves.

The limbs, since their motor fibres have decussated in the lower part of the medulla oblongata, are always palsied on the side opposite to the lesion of any level of the lateral half of the sensori-motor tract. In lesions of the medulla, near the decussation, however, there is rather a general weakness, and only hemiplegia in the qualified sense that the limbs are more affected on one side. As Brown-Séquard has pointed out in lesions of one lateral half of the medulla, the nerve-fibres from the limbs may be caught in part before and in part after their decussation, and thus from a one-sided lesion there results bilateral paralysis.

Above this great decussation, cranial nerves emerge from the sensori-motor tract, and many, if not the whole, of these nerves have special decussations. Hence in hemiplegia, parts supplied by the cranial nerves are sometimes palsied on the same side as the limbs, and sometimes on the opposite side, according as the lesion affects the fibres of these nerves after or before their decussations. For instance, both in lesion of the lower half of the right[1] side of the pons and in lesion of the right corpus striatum, the arm and leg are palsied on the left, but in the former case the face is palsied on the side of the lesion (the right), because the fibres of the facial (portio dura) nerve are caught before its decussation, whilst in the latter the face is palsied on the side opposite the lesion, because fibres of the nerve are caught after its decussation. We must observe further that the decussation[2] of cranial nerves, the facial and hypoglossal nerves at all events, is incomplete, so that, as we shall have to insist on later, we find in lesion of the higher levels of the motor tract, that is to say, in lesions above the pons, and therefore above the decussations of these nerves, that the palsies of the face and tongue are not only on the side opposite the lesion, but that they are incomplete in degree—there is paresis rather than paralysis.

CEREBRAL HEMISPHERE.—Large parts of one cerebral hemisphere may be destroyed without producing obvious symptoms, either mental or physical. It is not said that *disease* in the hemisphere does not *lead* to symptoms; it very often does. (See art. Convulsion, p. 281.) In the article on Convulsion it was pointed out that hemiplegia frequently attends gross disease, syphilitic disease for instance, of the surface of the hemisphere, but in these cases the hemiplegia nearly always follows a convulsion, and depends probably on the nervous discharge in the convulsion itself.

It is, we repeat, certain that a large quantity of any part of either hemisphere may be destroyed while there are no symptoms, *with one important exception.* If the lesion involves convolutions near the corpus striatum—usually the left—there is, according to the size and *exact* position of the lesion, more or less defect or even complete loss of speech (aphasia).[3] The probability is that when a part of the brain is *slowly* destroyed, the undamaged parts take on the function of the part destroyed. Something similar is seen in the case of the eye.

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[1] It will be convenient throughout this part of the article to suppose the right to be the side of the several lesions of which we have to speak.

[2] Lockhart Clarke has shown very clearly that there is a considerable decussation of the fibres of the ninth nerve. He finds that the nuclei of the facial are united by many fibres—commissural; but he has traced very few fibres of the *nerve* that decussate directly. (See Researches on the Intimate Structure of the Brain: Phil. Trans. 1868, p. 300, plate xiii. figs. 50 and 51.)

[3] This symptom is considered in art. Softening.

When central vision is lost, the peripheral parts of the retina acquire greater acuteness of vision. In large and *sudden* lesions of the cerebrum there are symptoms, probably from the withdrawal of a part before this kind of accommodation can be effected.

We may have no symptoms from small clots in the hemispheres. In very large hæmorrhages there are symptoms. These are usually, however, symptoms of Apoplexy; but if the Apoplexy be not very severe, we may discover some degree of hemiplegia. The palsy results either, as suggested, because a large part of the cerebrum is *suddenly* destroyed, or because the effusion by its mere bulk compresses the subjacent motor tract.

LATERAL VENTRICLES.—In cases where blood breaks into the lateral ventricle from the corpus striatum or thalamus opticus, there is usually, not always, a convulsion and rapidly deepening coma. The convulsion, however, I believe, occurs not because blood is effused in the ventricle, but because under such circumstances the clot is usually a large one and has *suddenly* torn much of the brain: for severe convulsion (with tongue-biting) may usher in the Apoplexy, or may occur later when a large clot, starting in the bodies mentioned, is well walled in. If a patient be first hemiplegic with or without unconsciousness, the subsequent occurrence of a severe convulsion followed by universal powerlessness and deep coma is strong evidence of rupture into the ventricle, and is a very unfavourable sign.

We now come to the sensori-motor tract. In the first two divisions—corpus striatum and thalamus,—the lesion is above the decussation, not only of the fibres for the limbs, but of all the cranial motor nerves, so that the paralysis produced is altogether on the opposite side of the body.

CORPUS STRIATUM.—A lesion here produces what may be called "the common form of hemiplegia." As this is the most valuable symptom in the diagnosis of Cerebral Hæmorrhage, it will be considered in some detail, but still only so far as bears on the diagnosis of Cerebral Hæmorrhage.

It is very important, however, to observe that a *very* large and sudden effusion in this region leads to such deep coma that, although there must be paralysis of the limbs, we often do not *discover* any (just as in deep coma with fracture of the base of the skull we may not discover palsy of the face due to injury of the portio dura nerve until the patient's coma is passing off). We discover no local palsy, because there is palsy of both sides—universal powerlessness. This may be the result of squeezing of the opposite side of the brain by the sudden intrusion of a bulky mass into one side, the blood perhaps having also escaped into the lateral ventricles. Possibly the universal palsy may be in part owing to the destruction—the *sudden* lack—of those fibres which pass from the side of the lesion to the *same* side of the cord, as well as of those which decussate. (I have heard Dr. Brown-Séquard insist that many hemiplegic patients, some time after their seizure, were a little weak on what we usually call their sound side.) However, even in these cases we may learn that the case *began* in a hemiplegic manner, or we find some kind of one-sided symptoms, as tremor, rigidity, and the like.

As we usually see hemiplegia, always in chronic cases and most often in recent cases, the palsy is *incomplete* in range. It affects only the face, tongue, leg, and arm of one side. But in *complete* hemiplegia we have the following symptoms. We suppose the right to be the side of the lesion :—

1. The head turns to the right.
2. *Both* eyes turn to the right, and frequently both upper lids are fallen.
3. The muscles of the belly and chest are weakened on the left.
4. The muscles passing from the trunk to the left limbs are paralysed.
5. The face is paralysed on the left side.
6. The tongue on protrusion turns to the *left.*
7. The left leg is paralysed.
8. The left arm is paralysed.

Hemiplegia so complete only occurs from a very grave[1] lesion, and even then, as a rule, the first two symptoms in the list pass off in a few hours or days. The symptoms in the list are given in what I believe to be the order in which the several parts suffer. It will be found that those parts suffer most and suffer longest (recover latest) which have the more voluntary uses. This is notorious of the arm and leg; the arm nearly always suffers more and recovers later than the leg. Of course the distinction into complete and incomplete hemiplegia is artificial. There are all degrees of paralysis according to degrees of gravity of the lesion. But there is an order in which paralysis increases in increasing gravity of lesions. We observe that the graver the lesion, not only are the more voluntary parts (arm and leg) *more* paralysed, but that the further spread in *range* is the paralysis, and the method of its spreading is from the more voluntary to the more automatic parts—to parts higher up in the list. Thus, neglecting very small clots, a considerable lesion (I cannot use a more exact term, suddenness of effusion as well as size of clot being a factor) paralyses only the most voluntary movements of one side of the body, those of the face, arm, and leg, and these parts in degree according to their degree of voluntary use. A larger lesion not only causes a deeper and more permanent palsy of these three parts, but it leads also to implication of more automatic parts; it causes the additional symptoms 1, 2, and 3 in the list. In still larger lesions the palsy spreads to the *most* automatic parts of the body, even to parts supplied by ganglionic nerves. It produces stertor from palsy of the palate and palsy of the respiratory muscles and of the heart—the palsy of respiration and of the heart showing itself chiefly in *slowness* of movement. There is also abasement of temperature. So we see that degrees of hemiplegia are "compound degrees." Not only are there degrees of more or less amount of loss of power of the face, arm, and leg—there is also, along with increasing degrees of loss of power of these most voluntary parts, increasing spreading of palsy to the more automatic parts of the body. There are degrees of hemiplegia, compound degrees as we have seen, from palsy of the most voluntary parts of one side only to almost universal paralysis, when, of course, "hemiplegia" is a misnomer.

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[1] This word is used to include two equally important factors, size and suddenness of lesion.

Lateral Deviation of the Eyes and Head.[1]—This is not strabismus, as both eyes are still parallel, although both are turned to the right side —to the side of the lesion; to the non-paralysed side. Indeed there never is, except as an accidental complication, strabismus from palsy of the third or of the sixth nerve in hemiplegia from a lesion above the crus cerebri. The patient,[2] especially when there is deviation of the head also, seems to be looking fixedly to one side. If the patient be sufficiently himself, we can get him to follow movements of our hand, and we may find that he can bring the two eyes to the middle line or even beyond it, but they soon fall again into the condition of deviation.

Along with this deviation there is often more or less turning of the head to the same side. There is, according to Vulpian and Prévost, "rotation," as there is after experimental lesion of one side of the brain in lower animals. But in man the rotation is only rudimentary; there is only a slight twist, not a real turning.

The clinical importance of these symptoms is that they may be, as Prévost suggests, valuable evidence of a local lesion—of a clot, for instance—in cases of coma where we can make out no paralysis of the limbs on one side, and when we are in doubt whether the coma is owing to a very large *local* lesion, such as extensive cerebral hæmorrhage, or to a general condition, such as uræmia or opium poisoning. Two things further are to be noted. In cases of convulsion of one side of the body, the two eyes and the head turn to the side convulsed, and they may be permanently turned to the paralysed side if it become rigid, and may strain still further in that direction when a convulsion or a higher wave of rigidity comes on. (Deviation of the head and eyes may occur in meningeal as well as in cerebral hæmorrhage.)

The Muscles of one side of the Chest.[3]—Whether the muscles of the chest suffer or not in this form of hemiplegia, is disputed. Niemeyer says: "Patients who, as a result of apoplexy on the left side, cannot move the right arm or leg, move the right side of the thorax just as well as the left during respiration." Todd says that "it must be an extensive lesion which will paralyse the intercostal and abdominal muscles." In some cases of recent hemiplegia, when the patient voluntarily draws a deep breath, the side of the chest paralysed certainly sometimes moves less than the other (see Wilks' Pathology of Nervous Diseases, Guy's Hospital Reports, 1866); probably, however, because the muscles passing from the trunk to the chest on the side of the paralysis will not act so strongly. At all events palsy of the muscles of one side of the chest is little, and soon passes off.

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[1] In this country attention was first called to this symptom by Gull, Lockhart Clarke, and Hutchinson. It has been described by Vulpian, and Prévost of Geneva (Gazette Hebdomadaire, Oct. 13, 1865). Cases by Drs. Humphry of Cambridge, Lockhart Clarke, Broadbent, and Russell Reynolds, and some interesting remarks on the symptom by Dr. Elizabeth Garrett (now Mrs. Garrett-Anderson), will be found in the *Lancet* for 1866.

[2] It has been likened to the conjugate deviation of the heads of two horses when an omnibus driver drops one of his reins, the other rein being "in tone." As, however, his reins do not decussate, that deviation is from the side of the "lesion."

[3] Dr. Broadbent has (Medico-Chir. Review, April 1866) advanced an important and very valuable hypothesis to explain how it is that whilst the muscles which can act

Face.—The muscles paralysed are those supplied by the facial (portio dura) nerve. The whole of these suffer, but they suffer slightly. Moreover we find that the several parts of the face do not suffer in the same degree. And it is to be particularly noted that there is only slight weakening of the orbicularis palpebrarum. The patient can close his eyes, although not so strongly on the paralysed side when urged to close them both tightly; sometimes, especially in chronic cases, we discover no difference. Hence this cerebral facial palsy differs remarkably from the facial palsy owing to affection of the trunk of the nerve (Bell's paralysis). The side of the cheek is the part most paralysed, so that when the patient is asked to "show his upper teeth" the mouth is decidedly drawn to the sound side.

The tongue is not paralysed; it is only weakened on one side, and on protrusion it turns but a very little to one side; it turns *to the side of the paralysis.* But sometimes, in cases of loss of speech with hemiplegia, especially soon after the attack, the tongue seems to be much paralysed, as the patient does not put it out when asked, even when he knows what is asked of him; he may try to get it out with his fingers. The fact that he can utter plainly some one or more words—mostly "yes" or "no"—that he masticates and swallows well, and that he may now and then put out his tongue to catch a stray crumb, shows that the tongue is not paralysed in the ordinary sense of the word. There is loss of the most voluntary movements of it, and we may find that the patient cannot do other simple things that he is told; *e.g.* open his mouth, shut his eyes, or frown.

Arm and Leg.—The arm suffers more and recovers later. We must not infer that the leg is not affected when the patient seems to move it as well as the other when he lies in bed. If we cannot get him up to walk, we can only say that the leg is not much paralysed. Indeed it is an exceedingly rare thing to find paralysis of the arm without *some* weakness of the leg soon after the attack, although not at all uncommon to find that the leg recovers when the arm remains much paralysed.

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quite independently of their fellows on the other side of the body (those of the arm, for instance), suffer in disease of the corpus striatum, those which must act together (the intercostals, for instance) do not suffer.

THALAMUS OPTICUS.—Disease of the thalamus opticus produces hemiplegia which, in regard of the motor symptoms, is very like that produced by disease of the corpus striatum. In grave lesions there is lateral deviation of the head and eyes. Probably, however, the palsy is not similarly distributed. I think, for instance, that the arm suffers less, and the leg more, the further back the lesion is placed in the two divisions of the motor tract, the corpus striatum and optic thalamus.

But in disease of the thalamus there is also diminution, or, soon after the attack, loss of sensation, and not of the arm and leg only, but of the whole half of the body, quite up to the middle line. This loss of sensation has been denied; one reason no doubt is, and it is important to bear this in mind, that sensation returns much more quickly than does power of movement.[1]

CRUS CEREBRI.—Damage to the crus cerebri causes hemiplegia on the opposite side like that above described, there being loss of motion if the under part of the crus only be damaged, and loss of sensation also if the upper strands be damaged as well. From the crus emerges the first cranial nerve, and at this level of the motor tract we may have "*cross paralysis.*" If the under and inner part of the crus be involved, the third nerve is paralysed on the side of the lesion—on the side opposite the paralysis of the limbs. The nerve is, so to speak, caught as it goes in,[2] and of course before any of its fibres can have decussated, whilst the fibres from the arm and leg are caught long after their much lower decussation in the medulla oblongata. But we can only make the diagnosis of lesion of the crus when the two symptoms—the palsy of the third nerve and the hemiplegia—come on at the *same time.* If they come on at different times, it is just as likely that there are two lesions, —one of the trunk of the nerve and the other in the thalamus or corpus striatum. This is important in the diagnosis of the *nature* of the lesion, for when the two symptoms—the palsy of the third nerve, and the hemiplegia—come out at different times, or if the palsy of the third nerve be on the same side as the hemiplegia, we may be practically certain that there is not Cerebral Hæmorrhage. In these cases the disease is mostly syphilis.[3]

So far the sides of the motor tract are distinct. In the next two divisions the two sides are welded together.

PONS VAROLII.—Hæmorrhage may be limited to one lateral half, or it may occupy both sides of the pons. (It may extend to the crus cerebri or crus cerebelli.) In the former case there is hemiplegia; in the latter double hemiplegia, or rather a condition of universal powerlessness, in which, as there is usually deep coma, we can make out no *local* paralysis—a condition, as we shall see, very like that of uræmia and opium poisoning. In other cases the palsy, although on both sides, is more marked on one side.

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[1] For important observations on this subject, see Broadbent, Med.-Chir. Review, April 1866.

[2] It is convenient to speak of the fibres of both the motor and sensory nerves as passing from their external distribution to the nervous centres.

[3] An important case of hæmorrhage into the crus cerebri has been published by Dr. Hermann Weber in the Med.-Chir. Society's Transactions, vol. xlvi. This report gives a careful account of the condition of sensation and of temperature on the two sides of the body; there is a reference to cases previously published.

We speak here of hemiplegia from lesion of one lateral half of the pons,[1] and we speak of lesions of the right half. Here again we have to do with cross paralysis, because we have to do with cranial nerves which decussate in the pons itself above the great decussation of the fibres for the limbs. The cranial nerves are the fifth, sixth, and facial. First, for general remarks on the effect of lesions at different levels of one lateral half of the pons, taking the facial nerve (portio dura) for illustration. In the right half of the pons there are fibres of the right and also of the left facial nerves, those of the right before its decussation, and those of the left after its decussation. (1) A lesion in the lower part of the pons will catch the fibres of the right facial nerve before its decussation, and will also involve the fibres from the left limbs, which have crossed lower down in the medulla. There is then palsy of the right side of the face, on the side of the lesion, and of the arm and leg on the left, the side opposite the lesion. (2) A lesion in the upper part of the pons (Brown-Séquard, op. cit. p. 153) may destroy fibres of the left facial nerve which have crossed in the pons, and will also involve the fibres for the left limbs. Here then the facial paralysis will be on the side opposite the lesion, and therefore on the same side as the palsy of the arm and leg. The hemiplegia will be like the hemiplegia from disease above the pons. Hemiplegia of this kind is very rarely caused by disease of the pons. (3) Lastly, Brown-Séquard points out, that a lesion in the middle of the pons will catch the fibres of the right facial nerve *before* its decussation, and those of the left *after* its decussation. Then the face is palsied on *both* sides from lesion of the right half of the pons and the arm and leg on the left.

To repeat, a lesion of the right half of the pons affecting the facial nerve near its implantation causes palsy of the face on the right side and of the arm and leg on the left. A lesion in the upper part of the right half of the pons causes the palsy of the face and of the limbs on the left side. A lesion of the right half of the pons may be so extensive as not only to affect the right facial nerve before its decussation, but to extend to the fibres of the left facial nerve after its decussation. Then there is palsy of the face on both sides, more marked on the right, and palsy of the left arm and leg.

What has been said applies to the fifth nerve. An extensive lesion may involve both the facial and fifth nerves, and then there is palsy of the region supplied by the fifth nerve and portio dura nerves of the right side and of the leg on the left.

Here it is to be observed that in most cases not only does the palsy of the face in cross paralysis from disease of the pons differ from the facial palsy in the common form of hemiplegia, in that it occurs on the side opposite the limbs paralysed, but it differs in that it is much more decided in degree. The facial palsy may be as extensive as that which results from disease of the trunk of the portio dura nerve. There is, in short, Bell's paralysis of the face. And when the fifth nerve is affected, the face is anæsthetic, the masseter and temporal muscles are much palsied, and after a time they waste as they do in disease of the *trunk* of the fifth.

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[1] In an article on Cerebral Hæmorrhage, we can only do so in outline. For a full account of the symptoms resulting from disease of the pons Varolii, see Brown-Séquard's Lectures in the *Lancet*, 1861, vol. ii.; and Lockhart Clarke's papers, Phil. Trans., 1868.

There are, in actual practice, combinations of symptoms which are more difficult to understand, but which can be resolved by a consideration of Lockhart Clarke's researches. (See especially Philosophical Transactions, Part I., 1868.) When the portio dura nerve is paralysed, we often find paralysis of the sixth nerve on the same side too. The opposite sixth may be also paralysed in a less degree (Brown-Séquard, op. cit.).[1] The relation of these two symptoms—the facial and ocular palsy—is easy to understand when we remember that the facial and sixth nerves, although they emerge at different places, arise, as Stilling and Lockhart Clarke have shown, from a common nucleus.

Again, we must observe that in limited disease in one-half of the pons, the parts supplied by the *motor* division of the fifth nerve may be paralysed on the side of the lesion when there is no diminution of *sensation* on that side. We find diminution of sensation on the *opposite* side of the face to that on which the limbs are affected. (This has occurred, in the cases I have seen, only when there has been palsy of the sixth and facial also.) Since Lockhart Clarke has found that the bundle of fibres of the sixth and the bundle of fibres of the portio dura nerve, after arising from their common nucleus, diverge so as to enclose the motor nucleus of the fifth—the fibres of the portio dura separating it from the sensory nucleus of the fifth—it is easy to understand why we have, from a single lesion, palsy of the portio dura, sixth, and the *motor division* of the fifth all on one side. But the occurrence of the diminution of sensation on the opposite side of the face is not easy to understand unless we assume a crossing of the sensory and motor fibres of the fifth in the pons, on separate levels, analogous to the crossing of sensory and motor fibres of the limbs on separate levels, which Brown-Séquard has discovered.

As to the limbs in hemiplegia from disease of the pons, there is usually affection both of sensation and motion. We find, of course, differences in the amount of loss of power, and of degree of anæsthesia. Moreover, we find differences in distribution of the two. I have seen the leg very much palsied and anæsthetic when the arm was scarcely weakened but nearly altogether anæsthetic. These points do not specially concern us in an article on Cerebral Hæmorrhage, although they are of very great physiological interest, as showing relations of strands of motor and sensory nerve-fibres for the limbs in the pons.

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[1] We should carefully examine the condition of the opposite sixth ; and if it be not markedly paralysed, we may see oscillations in the eyeball when the patient tries to avert it, showing that the external rectus on that side also is weakened.

Whilst the diagnosis of hemiplegia from hæmorrhage on one side of the pons is usually very easy, the diagnosis of hæmorrhage into both sides of the pons is sometimes very difficult. A large effusion in this part usually produces death rapidly—in a few hours. In large effusions there is usually marked contraction of the pupil on both sides, and there is universal powerlessness—a condition so like that of opium poisoning, that treatment for opium poisoning has been adopted in cases of hæmorrhage into the pons. (See Special Diagnosis.)

Medulla Oblongata.—Of the effects of effusion of blood limited to the medulla oblongata little is known. A large effusion would no doubt be very rapidly fatal, but I have never seen a large effusion here. I have made but one autopsy on a patient who had had a small effusion limited to the medulla. I saw this patient with Dr. Lockhart Clarke and Dr. Morell Mackenzie. The patient had recovered from an attack of hemiplegia due, as we afterwards found, to a clot in his right thalamus opticus, when all at once he lost power to articulate from paralysis of his tongue—remaining able to write well. A few years later the patient died, and Dr. Lockhart Clarke,[1] to whom I gave the medulla oblongata, found in it remains of past effusions of blood.

The symptoms which would lead us to infer disease of the medulla oblongata are paralysis of the lips, tongue, palate, and vocal cords. These symptoms, however, mostly come on very slowly, and usually both sides are equally affected. This is so in the "Paralysie labio-glosso-laryngée" of Duchenne. There can be no clot nor any kind of sudden lesion in such cases. If any of these palsies are on but one side, they must be attributed to tumour or to syphilis, if they come on slowly; but whether on one or on both sides, if they or any of them come on suddenly, they must be attributed either to clot or to softening from thrombosis.[2]

Cerebellum.—Nothing definite can be said as to the special symptoms produced by hæmorrhage into the cerebellum. Sometimes there is loss of consciousness, and sometimes there is not. Sometimes there is hemiplegia, and at other times none. And when there is hemiplegia, it is sometimes on the side of the lesion and sometimes on the opposite side. Of course, these differences depend on differences in the exact part of the cerebellum injured, on the size of the clot, and on the rapidity of the effusion, but these differences have not yet been put in order. When there are no paralytic symptoms, we can make no diagnosis. Sometimes there is a conjugate deviation of the eyes—not lateral deviation: one eye is turned upwards and outwards, and the other downwards and inwards. When this symptom is present, we may diagnose sudden lesion of the crus cerebelli. When there is hemiplegia, the palsy is not diagnostic as to the seat of the lesion unless perhaps we can ascertain the absence of facial and lingual palsy. For Brown-Séquard (*Lancet*, Nov. 2, 1861) says that in hemiplegia from hæmorrhage in the cerebellum there is neither lingual nor facial palsy, although there is a loss of facial expression. In cases of deep coma, however, we might be unable to tell whether there was facial or lingual palsy. But if there be hemiplegia of any kind in a case of apoplexy, we can at all events say there is a local lesion, and this with other symptoms would be good evidence of the existence of large Cerebral Hæmorrhage—and this is the most important matter—in some part of the encephalon. Vomiting occurs in Cerebral Hæmorrhage, but *very* urgent vomiting would point to Cerebellar Hæmorrhage: severe pain at the back of the head would supply still further evidence, but this symptom would only be presented when the patient was not unconscious. Fortunately, hæmorrhage in the cerebellum is very rare, so that we have not often the chance of being wrong.

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[1] See Phil. Trans., part i. 1868, where the case is published.

[2] Here I would refer the reader to very important remarks by Lockhart Clarke (Phil. Trans. part i. 1868, pp. 316–17, &c.), and to a case illustrating his views which I have published (London Hospital Reports, vol. i. 1864, p. 361).

THE APOPLECTIC CONDITION.

Apoplexy, as was remarked in the Introduction, p. 521, is not peculiar to Cerebral Hæmorrhage. It will be seen when we come to Special Diagnosis that it is sometimes difficult and occasionally impossible to tell whether Apoplexy is owing to a fatal lesion of the brain, or to the comparatively minor cause, deep drunkenness. Again, from clot there are all degrees, from slight and transient mental obscuration to profound and rapidly fatal coma. It is not *à priori* likely, when we consider that the clots vary in size, in the suddenness of their irruption, and in their seats of effusion, that there would be any uniformity in the conditions produced by Cerebral Hæmorrhage, and, as a matter of fact, the conditions vary very much indeed. We can here only speak of *severe* cases, admitting that they are not typical. Under the head of Diagnosis we shall notice cases of Cerebral Hæmorrhage in which there is no loss of consciousness; and under the head of Special Diagnosis, various degrees of impairment, or "loss of consciousness"—if the phrase *degrees* of *loss* of consciousness be permissible—will be spoken of.

The striking symptoms of the apoplectic condition are, (*a*) loss of consciousness, (*b*) states of pupil, (*c*) stertor, (*d*) alteration of pulse, respiration, and temperature.

Loss of Consciousness.—We are considering a severe case—apoplexy, the result of a large and sudden hæmorrhage—and as we admit that such a case is not typical, we shall only discuss how it happens that from a *circumscribed* lesion in but one side of the brain there results *total* abolition of consciousness.

We know that loss of consciousness cannot result from mere lack of the comparatively small part which the hæmorrhage has *destroyed*, for, as autopsies show, much of the brain may remain lacking for years in patients who have been unconscious only a few hours or days at the time when the destroying lesion occurred. But Cerebral Hæmorrhage is a bulky lesion—it squeezes; it is also a brusque lesion—it not only destroys, but it destroys suddenly—there is shock.

Niemeyer attributes the loss of consciousness to squeezing. He does not suppose it to depend on squeezing of the nerve-fibres and cells, but on squeezing of the capillaries; in other words, he attributes it to rapid anæmia of the brain, produced mechanically. It is true that there is no loss of consciousness in cases of very large cerebral tumour when there is evidently increased intracranial pressure; but clot and tumour are manifestly not comparable lesions. Tumours increase slowly, and probably, as Niemeyer suggests, the fibres of the part of the brain directly compressed become atrophous, and thus more room is made. Again, there is compensation by diminution in the quantity of cerebro-spinal fluid. But blood is rapidly effused, and the clot will squeeze before either of these modes of accommodation can take place, and thus the accommodation is, according to Niemeyer, obtained at the expense of the capillaries. They are emptied. In estimating the gravity of a lesion, *rapidity* is to be considered almost as important a factor as *size*, as will be best seen when we come to speak of sudden death from intracranial hæmorrhage.

Mr. Hutchinson (Lectures on Compression of the Brain, London Hospital Reports, vol. iv. 1867) also believes that the apoplectic condition is the result of *extensive* and *rapid* squeezing of a large quantity of the brain, and that the direct cause of the loss of consciousness is sudden anæmia *quoad* arterial blood. He points out that an enormous effusion, if it occurs very slowly, need not be attended by any insensibility whatever.

The above theory of the occurrence of loss of consciousness from anæmia, mechanically caused, may serve in the explanation of many cases of *large* Cerebral Hæmorrhage, but will not serve in all cases. It does not explain, as Jaccoud insists, the transitory loss of consciousness which sometimes occurs from small clots, the squeezing from which can be very trifling. Moreover, there are many cases of loss of consciousness from other causes in which there can be no squeezing, for instance in some cases of embolism[1] of the middle cerebral artery, and in the fit of epilepsy. In cases of laceration of the brain from injury —cases without any considerable effusion of blood being now considered—the apoplectic condition comes on although there is no squeezing. Thus Mr. Hutchinson says: "It is sometimes quite impossible to make any diagnosis by the symptoms alone [cases where there is no history of the mode of onset, for instance] between cases of sudden compression of the brain and those of laceration of the brain." We must consider the shock-producing element—that of suddenness of lesions.

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[1] He says, op. cit. p. 139, of the apoplexy which occasionally occurs from embolism, that it may be attributed "soit à une *névrolysie* produite par la perturbation subite de l'équilibre circulatoire (Jaccoud), soit à un œdéme aigu généralisé par suite de l'augmentation de pression dans les artères perméables (Niemeyer). Cette dernière interprétation me paraît difficilement admissible pour les cas où l'apoplexie dure à peine quelque minutes."

We may here avail ourselves of what Jaccoud has written in his important work, "Pathologie Interne," vol. i. p. 164. The normal function of the brain, he says, depends on the joint and simultaneous activity of its two halves. When one is injured, the other can in a certain measure *compensate,* provided the lesion occurs slowly, as in tumour. But the disturbance of a *sudden,* although local, lesion reacts on the whole brain, its two halves being *united by "powerful" commissures.* The torn brain receives a *shock* on the side injured directly, and this is transmitted and reflected on the other side, and then there is produced the "névrolysie"[1] which is apoplexy.

In unconsciousness produced by uræmia there may be no arterial anæmia in the ordinary sense of the expression, but still in effect the action will be the same. The blood is not good arterial blood. But, if we accept Traube's view that Bright's disease leads to œdema of the brain, there will be veritable anæmia in uræmia from the squeezing which the exuded fluid will cause.

Pupils.—There seems to be a wide-spread impression that when a patient is comatose, his pupils must be either "contracted" or "dilated."[2] It is quite certain that there may be a very large clot on one side of the brain when the pupils cannot be declared to be abnormal. Extreme contraction or extreme dilatation of the pupils are rare symptoms in Cerebral Hæmorrhage. It is to be observed that in the many cases of coma, although the pupils are very small when the patient is left still, as small as in healthy sleep, they may become much larger when attempts are made to rouse him. From not considering this, different accounts are given as to the condition of the pupils in the same case by different observers, or there are supposed to be remarkable variations in the signs of the pupils. On the whole, the conditions of the pupils are of little value.[3]

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[1] Niemeyer, op. cit. vol. ii. p. 184, admits that "the entire loss of consciousness, the apoplectic attack, which usually accompanies the commencement of hemiplegia when the arteria fossæ Sylvii is stopped by an embolus," is difficult to explain. He thinks it is "most probably due to the diseased hemisphere being decidedly swollen by collateral œdema, and that, as occurs in large extravasations of blood, the opposite hemisphere is not sufficiently protected from the pressure by the falx, which only offers a limited amount of resistance."

[2] In case-taking, I prefer the terms *small* and *large,* as they have not the misleading implications the more technical terms have.

[3] Dr. Wilks says (Guy's Hospital Reports, 1866, p. 177) that "we cannot connect the conditions of the pupils with any definite lesions, for their state is very variable and liable to be influenced by very slight causes." Speaking of the contraction of the pupils observed in disease of the pons, he adds, "just as we see this produced by effusion of blood at the base or into the ventricle." Callender (St. Bartholomew's Hospital Reports, vol. iii. p. 430), speaking of the pupils in cases of injury to the head, says, "their condition varies remarkably in these cases, and no sort of reliance can be placed upon the appearance they may seem to present."

We shall speak of contraction of both pupils under the head of Diagnosis of Apoplexy, owing to hæmorrhage into the pons, from opium poisoning. The following further remarks on the pupils belong strictly to diagnosis, but they are most conveniently considered here.

Importance must be attached to difference in the size of the two pupils, but only when the difference is great; for the pupils are often of slightly different size in healthy people. Difference in the size of the pupils points to a local lesion. Thus, were one pupil very minutely contracted and the other presumably unaltered, the contraction would be some evidence of disease of the pons Varolii on the side of the contraction. But I have not yet seen minute contraction of the pupil on one side from clot in the pons when there has been coma. I have seen it in cases of hemiplegia from disease of one side of the pons: in one of these cases there was a clot. Were one pupil *very widely dilated*, it would lead me to search most carefully for signs of injury to the head, as Mr. Hutchinson has found very wide dilatation of one pupil in cases of blood effused under the dura mater in the sphenoidal fossa from fractured base; the dilatation is on the side of the effusion.

During convulsive seizures the pupils may dilate very widely; this is not always the case.

Stertor is the sign of a grave lesion, or more generally of serious implication of the brain, and, like the other symptoms of the apoplectic condition, is not of diagnostic value. There may be uræmia, alcoholic poisoning, or large Cerebral Hæmorrhage. It will occur in any one of these conditions. It may be absent in any of them, and is often absent in apoplexy from Cerebral Hæmorrhage. The noise made will depend on the condition of the respiration, and varies with it in the same case. It shows that the lesion is large enough or widely enough spread in the brain to affect muscles supplied by or through the ganglionic nervous system. It is of value in prognosis. It shows grave lesion.

Pulse, respiration, and temperature.—This is the most important and at the same time the most difficult part of the subject. It is really impossible to give a proper account of the condition of pulse, respiration, and temperature without citing numerous cases, and these would show extreme differences, differences so great that it is most difficult to make generalizations. The great point to observe is that the condition of the pulse, respiration, and temperature varies very much according to the time elapsed from the seizure. So that so far as the pulse, respiration, and temperature are concerned, the patient may be in opposite conditions according as he is seen early or late. And since the lesions differ greatly in gravity—in size and suddenness—we can say nothing definitely as to time. What is early in one case is late in another. This is obvious when we consider that a clot may destroy life in an hour, or may not kill for days, or that the patient may recover from it. Moreover, alterations of pulse, respiration, and temperature, depression of them at all events, are not peculiar to Cerebral Hæmorrhage; they are found in cases of meningeal hæmorrhage, alcoholic poisoning, and even in some cases of tumour of the brain. We are obliged then to speak most generally. For convenience we make two stages of the apoplectic condition: one we call collapse and the other reaction. There is no absolute demarcation betwixt the two. Loss of consciousness continues through both. Of course, if the clot be small there are no stages—neither collapse nor reaction. Again the patient may recover from his collapse, if it be faintly marked, without any obvious reaction; he may die rapidly, and then there is practically no question of stages.

First stage:—Soon after the effusion, even of a large clot, the pulse, respiration, and temperature may be absolutely normal, the patient seeming as if gently asleep. But we shall speak of cases in which they *are* abnormal; the symptoms are those of depression. The face is pale, the pulse is slow and laboured, the respiration is shallow, and the temperature is lowered. The pulse may be 60 or under, the temperature may be in the axilla 96 or lower.

In the second stage, the stage of reaction, the pulse quickens, respiration quickens, and the temperature usually rises, for instance, to 101, or 103. Shortly before death, it may rise to 107 or more. These points concern us most in prognosis: the quicker the pulse and respiration, and the higher the temperature, the less likely is the patient to recover. We frequently observe also that the pulse becomes irregular or intermittent. The respiratory action undergoes great variation in frequency. Thus the patient for a while lies breathing quickly, but pretty evenly in rate, and then for a short time there is a series of more rapid respirations, with loud stertor, after which comes a period of comparative calm.

Again, not only is the rate of respiration to be considered, but the character of the respiratory movements are to be noted. As they quicken in *rate*, so do they become more extensive in *range*, though each respiration is still short. Thus, in the first stage there may be only quiet action of the diaphragm, but at length the sides of the chest evert strongly in inspiration, the abdominal movement being less obvious, and at length the upper thorax takes part in the process. In severe cases the epigastrium sinks in during inspiration. This is probably partly owing to elevation of the attachments of the diaphragm from increased action of the sides of the thorax, and partly to pushing down of the diaphragm by the increasing bulk of the lungs from congestion or œdema.

DIAGNOSIS.

An account has been given of the general bodily state of the patient who is especially liable to Cerebral Hæmorrhage. We have described the symptoms which we know that hæmorrhage produces—both the local, such as varieties of hemiplegia, and the general, the apoplexy which occurs from large and sudden hæmorrhages; but other lesions produce exactly similar local symptoms, and many conditions cause Apoplexy. We now come to the most difficult part of our subject—Diagnosis. Under this head we speak first of Premonitory Symptoms; next of Modes of Onset, under which will be considered the comparatively simple cases of patients who have hemiplegic symptoms; and lastly, under Special Diagnosis, of the condition of those apoplectic patients in whom we can discover no hemiplegia. The separate consideration of those cases of Cerebral Hæmorrhage in which there is, and of those in which there is not hemiplegia, is justified by convenience; for practically they are different things. When called to a patient who is hemiplegic, whatever other difficulties we have, we are certain, since there is clearly a *local* lesion of some kind, that there is not only drunkenness, uræmia, poisoning, &c.; but if we make out no hemiplegia, we may be in doubt whether the apoplexy be owing to a very large clot in the brain, to a very central one as in the pons, or to some one of the more general conditions mentioned.

PREMONITORY SYMPTOMS.—The general bodily condition already described (p. 527 et seq.) furnishes the best basis for premonition, and in no nervous affection in people of, or past, middle life, however trivial the symptoms may be, do we neglect to examine the heart, arteries, and urine; but that condition leads to, or is associated with, disease of many parts of the body. We here speak of slight *nervous* symptoms which point more expressly to the future occurrence of Cerebral Hæmorrhage, and which, when occurring in a patient who has degenerated arteries, hypertrophy of the left ventricle of the heart, and chronic renal disease, show it to be very likely that this unsound state in his particular case is about to lead to decided hemiplegia, or fatal apoplexy, from the rupture of vessels in the nervous centres. Some of these patients, however, die after having had slight warning symptoms,—some evidently due to hæmorrhage, for we may have seen clots in retinæ and have heard of epistaxis—in other modes, as by pericarditis, uræmia, &c.

Before we speak of special premonitory symptoms, we must remark that some patients who die of Cerebral Hæmorrhage have had none, at least we hear of none; and this is sometimes the case when examination both during life and after death reveals signs of most extreme degeneration.

The degeneration of arteries of which we have spoken (p. 529) leads to two pathological states of nervous centres: to softening by thrombosis, and to hæmorrhage by rupture. The slight symptoms of which we are about to speak as premonitory of Cerebral Hæmorrhage may depend on either; practically it matters little, since the symptoms to be mentioned may be taken as warnings of the possible supervention of *large* Cerebral Hæmorrhage, whether they signify small hæmorrhages or very limited softening. It may, however, be denied that there is any local lesion when such slight symptoms as those I have to mention pass away altogether in a few hours or days. Besides the reply that local symptoms of necessity imply local lesion, it may be added that it is quite certain that even decided hemiplegia, whether from clot or from softening, will pass away even when, as subsequent post-mortem examination shows, there remains a permanent, although a small, void in the motor tract.

One general remark may be made: the premonitory symptoms of Cerebral Hæmorrhage are owing to affections of nervous centres, and not of nerve-trunks. In cases of Apoplexy from rupture of aneurisms of the larger cerebral arteries, there may have been palsy of a nerve-trunk—the third especially—from compression of that nerve-trunk by the aneurism; but rupture of such aneurisms, with very rare exceptions, leads to meningeal, not to cerebral, hæmorrhage. There are, however, some seeming exceptions to the rule laid down. We may have palsy of the tongue from hæmorrhage into the medulla oblongata, and palsy of parts supplied by the facial nerve from thrombosis or small clot in the pons;[1] but these are, as stated, only seemingly exceptional. They are *not* owing to affections of nerve-trunks; and practically, when we are consulted for palsy of any cranial nerve, we do not attach much importance to it as a warning of Cerebral Hæmorrhage, we think of syphilis, and, if the nerve palsied be one of those to the muscles of the eyeball, of locomotor ataxy also.

So far negatively. The symptoms which are premonitory of Cerebral Hæmorrhage are innumerable. We may divide them into two classes, local and general. The local ones to be mentioned are:

Defect of Sight[2] occurs now and then before Cerebral Hæmorrhage. In these cases we find mostly the degeneration of the retinæ which occurs with Bright's disease, and usually linear clots are to be seen in the retinæ too; indeed it is an affection of a nerve centre,[3] and of one supplied by the same arterial system of the brain, and is not owing to affection of a nerve-trunk. The existence of these changes makes us take a very gloomy view of the case of a patient who has even the slightest nervous symptom.[4] If we find either optic neuritis or any kind of optic atrophy, we cannot infer liability to Cerebral Hæmorrhage unless we know that the atrophy has followed the neuro-retinitis of Bright's disease. Indeed optic neuritis (there being no albuminuria) is rarely associated in any way with Cerebral Hæmorrhage. It rarely precedes, and it very rarely follows it. As, however, optic neuritis is frequently associated, especially in young persons, with tumour of the brain, there is to be considered the liability to hæmorrhage from tumour; but this is of very rare occurrence.

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[1] Dr. Moxon has recorded (Path. Soc. Transactions, 1869–70) a case of paralysis of the portio dura nerve from hæmorrhage into the aqueductus Fallopii.

[2] I do not know that deafness is of value as a warning of Cerebral Hæmorrhage; it is a rare symptom in cases of serious brain disease of any kind.

[3] In many of these cases of retinal hæmorrhages there has been nasal hæmorrhage also. Epistaxis, however unimportant in itself, is a serious warning if there be chronic Bright's disease. It is of very great importance to use the ophthalmoscope in all cases of brain disease. There are often changes significant of Bright's disease, so well marked that the ophthalmic surgeon is the first to discover that the patient has Bright's disease. *There may be no impairment of sight* when the ophthalmoscopical appearances are extremely well marked. This is to be strongly insisted on.

[4] I may here refer to a record of several cases in a lecture on Cerebral Hæmorrhage (Lond. Hosp. Reports, vol. iii. 1866). In some cases of coma the detection of these changes may enable us to make the diagnosis of Cerebral Hæmorrhage.

Limited Facial Palsy.[1]—This is really a part of an attack of hemiplegia. It is the kind of facial palsy which occurs in hemiplegia which is so common in disease of the higher motor track. (See p. 539.) We must observe, however, that a facial palsy of exactly the same kind occurs after certain epileptiform seizures; but in these cases there is occasional spasm of the paralysed part. The patient is usually young, and we have often a history of syphilis. The facial palsy which is a warning of Cerebral Hæmorrhage comes on suddenly without spasm, or is found on waking, and usually passes off in a few days. It is a very unfavourable sign, because it shows *central* disease.

Speech.—There may be *loss* of speech with the above-mentioned kind of facial palsy, but more often there is *defect* of speech only; a difficulty of articulation for which the degree of palsy of the face and tongue does not account; and we find that the patient writes, that is expresses himself in writing, about as badly as he talks. Again, there is central disease. Speech defects are not of special value as warnings of Cerebral Hæmorrhage unless they come on suddenly. We must also bear in mind that temporary loss of speech occurs from embolism; at all events it occurs in young patients who have valvular disease of the heart. Occasional mistakes in words occur in many presumably healthy people, and their significance as evidence of coming cerebral disease of any kind is, I think, overrated. Suddenly occurring difficulty of articulation is the condition of most evil import.

Slight weakness or numbness of one arm and leg points to a local and central lesion, although to a minute one; but many people are subject for years to a slight numbness and to queer feelings on one side, who seem to be otherwise in good health, or the symptom obtrudes itself when they are slightly out of health—dyspeptic, for instance. It is not uncommon in young and apparently healthy people. A slight weakness of one side is only of value as a warning of Cerebral Hæmorrhage when it occurs suddenly without spasm, and even then we do not attach great importance to it unless the patient be past middle age and show signs of degeneration.

Successions of slight local symptoms are of more value than any single symptoms. From syphilis also there are successions of nervous symptoms: they are such as palsies of cranial nerves, optic neuritis, partial convulsion. But preceding fatal Cerebral Hæmorrhages we may find epistaxis; defect of sight from degeneration of, often with clots in, the retinæ; sudden numbness or weakness on one side (without spasm); occasional difficulty of articulation, and drawing of the face. These are due to affections of nerve-centres; or the patient may have many epileptiform paroxysms of very different kinds, sometimes slight without loss of consciousness, sometimes severe with tongue-biting, sometimes local, sometimes general. Here we suspect small meningeal hæmorrhages.

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[1] See Trousseau (Bazire's Trans.), vol. i. lect. 1.

General Premonitory Symptoms.—A page might be filled by the enumeration of symptoms of this class which authors give as warnings of Cerebral Hæmorrhage. They are such as drowsiness, loss of memory, especially for recent events, irritability of temper. Such symptoms point only to general deterioration of brain, to slow wasting, for instance—and do not point especially to a liability to Cerebral Hæmorrhage. More valuable symptoms of this class are giddiness, pain in the head, and vomiting. But these again may be found in the onset of many kinds of brain disease; for instance, in young people the subjects of cerebral tumour: indeed, if the headache be intense and continued for weeks, and if the vomiting be urgent, it is, provided there be no albuminuria, likely that there is tumour. If, however, the symptoms come on suddenly in a person of middle age, especially if there be slight confusion of mind at the time, and above all if there be any paralytic symptom, however faintly marked, such as thick speech or unilateral weakness, they may be taken as warnings of Cerebral Hæmorrhage. Doubtless they are owing to small clots or to limited thrombosis. If there be albuminuria, they are often ascribed to uræmia. Patients with chronic Bright's disease are prone to attacks of headache and vomiting, especially on getting up in the morning. Even if these symptoms are dependent on uræmia, they may still be considered as warnings—indirect, it is true—of the possible future advent of Cerebral Hæmorrhage, when they occur in a person past middle age who has tough arteries, hypertrophy of the left ventricle of the heart, and no notable dropsy with his albuminuria.

THE MODE OF ONSET OF CEREBRAL HÆMORRHAGE.—This is often the only diagnostic evidence of value, and in many cases when it is not forthcoming we cannot make a diagnosis at all—as when a patient is found by the police in the streets "drunk and incapable."

When we consider that the clot differs in seat, in size, and rapidity of effusion, we cannot *à priori* expect any great uniformity of manner of onset; as a matter of fact it varies very greatly indeed. A patient may not be unconscious from a hæmorrhage large enough to produce permanent hemiplegia, or he may, minutes or hours after being hemiplegic, become apoplectic, or he may become apoplectic almost without prior symptoms. I say *almost*, because however quickly Apoplexy from Cerebral Hæmorrhage comes on, there are nearly always *some* prior symptoms. As Trousseau says, Apoplexy, in the classical sense of the word—a sudden falling—is rare in Cerebral Hæmorrhage. There is nearly always *something* wrong before the patient becomes unconscious, and often the interval is considerable—minutes, or even hours. Trousseau excepts cases beginning by convulsion, and also cases of hæmorrhage into the pons Varolii; but even in cases of hæmorrhage into the pons, there are, I believe, mostly some symptoms before the loss of consciousness. Thus, I have notes of a case of a man who came off a scaffold because he was giddy before he became insensible, and of another patient who when taking a drink cried out that he was poisoned. Dr. Hare relates the case of a patient who was able to knock at a door and say she was going to die before she became insensible. In each of these cases there was large hæmorrhage into the pons. In considering special modes of onset we shall give further illustrations. The first statement as to mode of onset is that Cerebral Hæmorrhage, even when large enough to produce Apoplexy, does not as a rule cause Apoplexy instantly.

To consider mode of onset more particularly we must make a grouping of cases. The following, although in actual practice we see all degrees of intermediateness, is convenient.

Rapid Death.—This practically includes onset and termination. It is a rare thing for Cerebral Hæmorrhage to cause rapid death; within half an hour, for instance. From theoretical considerations we might suppose that when the clot is effused near to the medulla oblongata—in the pons—death would occur very rapidly; but as a matter of fact it rarely does. Yet cases of sudden death are frequently put down to "Apoplexy," Cerebral Hæmorrhage being meant. Since this term is often made to include meningeal hæmorrhage, the statement is not altogether wrong, for meningeal hæmorrhage may cause death in a few minutes—five, for instance, and probably in less time. Yet, since meningeal hæmorrhage may lead to death very slowly, we have to infer that it is *rapidity* of effusion which is rapidly fatal, and not the position of the hæmorrhage. If the patient dies rapidly, within half an hour let us say, *cerebral* hæmorrhage is most unlikely. If he be young and healthy-looking, the probability is that there is *meningeal* hæmorrhage from rupture of an aneurism of a large cerebral vessel. Usually there is a convulsion in these cases, and if death occurs "in a fit," or very quickly after, we still incline in a young person to the diagnosis of ruptured aneurism; but it is quite certain that in some cases of death in a first convulsion we discover nothing abnormal post-mortem. If death occurs instantly—the patient dying in a minute—we infer failure of the heart, rupture of aneurism of the aorta into the pericardium, or rupture of the heart itself. Death by intracranial hæmorrhage is never so exceedingly rapid as it often is from these causes.

Convulsion.—This mode of onset has been considered in the article Convulsion, p. 252. What further is to be said will find its place best when the cases of patients who are apoplectic without discoverable paralysis are spoken of. However, convulsion from clot is frequently followed by apoplexy with hemiplegia; such cases will be considered under the head of "Hemiplegia with loss of consciousness."

Hemiplegia without loss of Consciousness.—If we are called to a patient of, or past, middle age who is hemiplegic without loss of consciousness, we have to consider two possible kinds of lesions:

softening from thrombosis;[1] clot from rupture of a vessel. In the great majority of cases hemiplegia without loss of consciousness is the result of local softening. I believe we can say very little more than this, for a clot occasionally causes permanent hemiplegia without producing loss of consciousness at its irruption. However, a very deliberate mode of onset strongly favours the diagnosis of softening. If, for instance, a patient, when he gets up in the morning, finds his arm weak, next his leg numb, and half an hour later is paralysed on one side, little or much, we diagnose softening. What has been said before as to constitutional state—or as to premonitory symptoms—helps us but little in this difficulty, for degeneration of arteries leads either to softening or to clot, and any local premonitory symptoms the patient may have had may have been the result either of thrombosis or of rupture of small arteries. Still, the existence of chronic Bright's disease is much in favour of clot; and if we see clots in the retinæ and hear that the patient has had epistaxis, we are warranted in inferring from these visible hæmorrhages that the encephalic lesion is also hæmorrhagic.

When the hemiplegia is on the right side, and now and then, but very rarely, when on the left, there is loss or defect of speech. This furnishes no further diagnostic evidence. I think, however, that frequent mistakes in words during recovery in young[2] people favours the diagnosis of plugging of vessels.

Hemiplegia with Loss of Consciousness.—Hemiplegia with deep loss of consciousness (Apoplexy) is nearly always owing to Cerebral Hæmorrhage. These cases are therefore more important than any other, and we must consider the mode of onset in some detail. The attack may begin either by special nervous symptoms, such as one-sided numbness, loss of speech, defect of speech, or by such symptoms as pain in the head, vomiting, and confusion of mind, and, of course, it may begin by both sorts of symptoms at once. It may begin by convulsion. We here consider the special symptoms only; the general symptoms are of additional diagnostic value, but we could only repeat what has been said (p. 552) when speaking of them as premonitory symptoms.

When a patient has suddenly, decided, although very slight, local palsy (for instance a little thickness of speech, a trifling drawing of the face, or loss of use of one arm, one-sided weakness, or even numbness), it is clear enough that he has some kind of *local* lesion of his nervous system. We should believe hæmorrhage was that lesion if any one of these symptoms were followed quickly by deep loss of consciousness, or if after some deliberation, or even if after partial recovery, a convulsion occurred. If the patient be above middle age, if he have tough arteries, if there be albuminuria, we are almost certain that hæmorrhage has occurred; and if, after such a mode of onset, hemiplegia is found with deep coma, we are practically quite certain. If the mode of onset has been by convulsion, we still think it most likely that there is hæmorrhage, if there be decided, and especially if there be *complete* hemiplegia—complete *in range,* that is. (See list p. 537.[1])

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[1] For further points in diagnosis, and especially for the diagnosis of softening from embolism, see art. Softening.

[2] Plugging of cerebral arteries in older people is not the same thing as plugging of cerebral arteries in young patients, as the vascular condition of the brain is different at different ages. There is in older people less free anastomosis from obliteration of capillaries, and also from the atheromatous condition of small arteries. The vessels of the optic disc become fewer in number as age advances.

We now consider the case of a hemiplegic patient when he is fully apoplectic. As before said, the degree of the coma in cases of Cerebral Hæmorrhage varies; the deeper it is, the more is the diagnosis of hæmorrhage warranted: but loss of consciousness, accompanied by stertor, slow pulse, lower temperature, is not diagnostic of Cerebral Hæmorrhage. If we have no history of the mode of onset, or only that the patient was taken with a fit of convulsions, the first thing we do is to inquire for hemiplegic symptoms. While hemiplegia is certain evidence of the existence of local lesion, and with other circumstances of the existence of clot, we must not suppose that its absence negatives clot. (See p. 536.) Hence we have often difficulty in saying whether there is erebral æmorrhage, or poisoning, or uræmia. We usually discover some kind of one-sided symptoms if we do not find definite hemiplegia; we find *some* difference in the two sides when we raise the arms and let them fall, and when we pinch the legs. There may be spasm, or there occur occasional waves of tremor down one side; or we may find both eyes or the head turned strongly to *one side.* These symptoms point decisively to a one-sided lesion at all events; and when there is no history of injury, no evidence of embolism,[2] they mostly, in persons past middle age, signify clot. Yet there may be meningeal hæmorrhage, and if the limbs of one side be continuously rigid, with or without occasional higher waves of rigidity, the probability is that there is meningeal hæmorrhage, although perhaps cerebral as well. However, and this really is the important matter, very deep coma occurring suddenly or quickly with *one-sided* symptoms of any kind, point at least to intracranial, if not to cerebral, hæmorrhage, in the vast majority of cases of patients past middle age.

SPECIAL DIAGNOSIS.

Apoplexy without Local Paralysis.—When there are no local symptoms in the apoplectic condition, no hemiplegia for instance, it is most difficult to make a diagnosis. We shall here discuss only the difficulties we most frequently encounter. We shall suppose that we are called to a case of coma, and try to show by what means we may arrive at the diagnosis of Cerebral Hæmorrhage. We often cannot; but even then we may be able, at all events, to decide whether there is a fatal lesion, or the comparatively minor condition, drunkenness, and to exclude violence and poisoning. Speaking generally, the difficulty is to determine whether a patient is suffering from *local* lesion so large and sudden, or placed so centrally (as in the pons), as to produce coma with universal powerlessness, or whether he is suffering from some condition such as uræmia, poisoning by opium, drunkenness, &c., which, as it were, imitate the effects of the grave local lesions mentioned.

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[1] See, however, art. Convulsion, p. 279, "Epileptic Hemiplegia."

[2] See art. Softening.

It is important to bear in mind that we may have combinations of states. I have known an "epileptic" fracture his skull by a fall in a fit, and die from hæmorrhage the result of rupture of the middle meningeal artery. A drunken man may have been struck on the head. A drunken man falls like a log, and a seemingly slight blow on the kerbstone, for instance, will lead to hæmorrhage into the arachnoid cavity. I have seen two cases of this kind in drunken people. Prescott Hewett says that extravasation of blood in the arachnoid cavity is much commoner than is usually supposed; that the injury causing such extravasation is often a trifling one; that it may occur without any apparent lesion of the brain or membranes. Mr. Stephen Mackenzie has known a patient who died of garotting to be treated for alcoholic poisoning; the patient *was* drunk when attacked. If the patient be often drunk, a fit of drunkenness may not improbably coincide with, and perhaps be the direct cause of, rupture of cerebral arteries.

But the difficulty is not nearly so great practically as it is logically; for when we know the constitutional history of an apoplectic patient (see p. 527), and if we are told, as we mostly are, the mode of onset (see p. 552), we are very rarely in doubt as to the cause of Apoplexy. We may know that the patient has had an attack of hemiplegia or some other paralytic symptom (see p. 549) before; and this will favour very strongly the diagnosis of hæmorrhage. Then cases of Apoplexy without hemiplegia or without some hemiplegic symptom are comparatively rare.

In most cases the patient becomes ill, at home among his friends, or at his work, or he is found comatose in bed, and in the great majority of instances the *circumstances* negative drunkenness, poisoning, violence, and the like, when the symptoms do not. Indeed, in most cases the diagnosis is really easy; or, to speak strictly, the prediction is usually verified. Suppose, however, there is no history. Suppose the patient, as is pretty often the case in hospital practice, is found in the streets universally powerless and deeply comatose, we very often *cannot tell from what he is suffering.* Or let us suppose we are called to a guest at an inn, who is found comatose in bed or in the water-closet—the discovery of an empty laudanum bottle may be the only clue to the nature of the case. In such cases we can only say there is apoplexy; we cannot declare the cause of it, and simply because there is not evidence. I would most earnestly beg young practitioners not to trust blindly to the fact that the patient is found at the bottom of a scaffold in the diagnosis of injury, nor to the smell of drink, nor to an "uproarious condition," for the diagnosis of intoxication;

and, above all, not to conclude, from bitten tongue, that the patient has "only had an epileptic fit." It is true enough that if he be led entirely by these circumstances, he will *mostly* be right, but he must run no risk of being wrong. Most painful mistakes are occasionally made because a practitioner concludes from insufficient evidence.

Having first examined the apoplectic patient for hemiplegia (the existence of which we are now supposing that we cannot determine), we next inquire for convulsions. If we obtain no history of a convulsion, we search for evidence of tongue-biting. However, we cannot often get a look at the tongue, but we may judge by the presence of blood on the gums or by bloody foam. If the foam be very frothy in large bubbles, it no doubt comes from the bronchial tubes, and is no evidence of tongue-biting. The tongue may have been bitten but not lacerated. And when we do find evidence of convulsion, we must remember that to use the words of Gull,[1] "*general* convulsion with insensibility is in itself of *little value* in the diagnosis of *any* brain disease." (The italics are mine.) If we feel sure that there has been a convulsion either before or after the onset of the symptoms, we can only exclude drunkenness and poisoning. If there be no convulsion, we have still these two causes to consider. We next examine the urine for albumen. We speak first of the comatose patient who is not hemiplegic and who has had no convulsion, so far as we can tell, and whose urine is not albuminous. The first question is,—

Drunkenness.[2]—The smell of drink must only lead us to a very careful examination for evidence of drunkenness, as patients who suffer Cerebral Hæmorrhage may have been drinking, or may have taken spirits for premonitory symptoms. Oddly enough, patients soundly drunk, their real condition not being recognised, are now and then treated by doses of brandy-and-water. This shows in another way the difficulties of diagnosis. A drunken man may be in one of two conditions. (1) He may be insensible without excitement; he may, indeed, be as deeply comatose as if he had extensive and fatal Cerebral Hæmorrhage. This is so when the patient has been "sucking the monkey," *i.e.* sucking raw spirits out of a cask by aid of a gas piping, or when he has drunk off a large quantity of spirits for a wager or out of bravado. In these cases, from the condition of the patient alone we cannot make a diagnosis, although, fortunately, it is usually made for us by the history. If we hear that the insensibility came on very slowly while the patient was drinking, especially if it were preceded by excitement of talk or manner, we should suppose we had to do with drunkenness. If, however, the insensibility began suddenly, or if there were a sudden increase of stupidity, or if the patient all at once staggered and fell insensible, cerebral or meningeal hæmorrhage is almost as likely. Let us now suppose there is no history of the mode of onset, the patient being found in the streets by the police. We try to rouse him, and we may get him to give his name or his address. This is, perhaps, some evidence that the case is not one of Cerebral Hæmorrhage, but it had better be disregarded, as patients comatose from fatal cerebral lesions of several kinds can be roused so far. That he resists our endeavours to examine him, or swears when roused, is of no value at all as excluding fatal lesion of the brain. The patient may vomit (as he may in Cerebral Hæmorrhage), and the vomit may reveal the nature of the case. If he does not we are justified, in doubtful cases, in using the stomach-pump. Then, the drunken patient oftener passes his urine and fæces than do other apoplectic patients. Again, we may find alcohol in the urine. The mere presence of alcohol in the urine is not to be relied on to show that the apoplectic patient is suffering from a *poisonous* dose of alcohol only. As before said, a drunken man may owe his coma, in part at least, to hæmorrhage into the arachnoid cavity. However, Dr. Anstie tells me that it would be possible to recognise the presence of a *poisonous* dose of alcohol in the system if one drop of the urine itself, added to 15 minims of the chromic acid solution,[1] turned the latter *immediately* to a bright *emerald* green.

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[1] Abscess of Brain, Guy's Hosp. Reports, vol. iii. third series.

[2] Here I would refer to papers on Alcohol Poisoning by my colleague, Dr. Bathurst Woodman, in the *Medical Mirror*, July 1865 and February 1866. Dr. Woodman has had an unusually large experience of cases of apoplexy from numerous causes, and to him I have to acknowledge myself greatly indebted for facts serving in the diagnosis of causes of coma. I have recorded (Lond. Hosp. Rep. vol. i. p. 35, from notes by Dr. Woodman) a case of death by hæmorrhage in the pons, in which, when the patient was first seen, the symptoms were like those in some cases of deep drunkenness. He could move all his limbs, put out his tongue when asked, and, although insensible, was roused by shouting to answer, "What's that to you?" when asked his name. He had been found in the street by a policeman.

(2) The other condition is one of excitement, of which there are all degrees; as we have seen, the patient, who when left to himself is insensible, may be roused to resist and to swear, but the main features of a case to which we are called may be one of "uproariousness." If the patient be violent, and struggle, he is probably drunk. A cautious man will still continue his examination for other causes, because it is certain that after severe and fatal[2] injuries to the head the patient may struggle and swear, and even, as I saw in one of Mr. Hutchinson's cases, make replies as definite as "What's that to you about my tongue?" when asked to put his tongue out. I have recorded a case supplied to me by Mr. Stephen Mackenzie,[3] in which violence and swearing were the striking symptoms in a case of death from meningeal hæmorrhage. As in this case, we have often a history of a mode of onset under circumstances which exclude the diagnosis

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[1] The chromic acid solution is made by dissolving one part of bichromate of potash in three hundred parts by weight of strong sulphuric acid. Of course Dr. Anstie does not represent this test as a certain one for alcohol, but there is not likely, he tells me —and his experience on this point is very great indeed—to be any practical objection to the conclusion when the reaction is so sudden and decided on the addition of but a drop of urine to the test solution.

[2] See Callender, St. Bartholomew's Hosp. Rep., vol. iii. p. 415, and especially Case 5 of his series of cases.

[3] Medical Times and Gazette, April 1, 1870.

of drunkenness. But to make a diagnosis from the condition of the patient only is quite a different thing. We can only make a diagnosis by exclusion, and the most important thing is to exclude injury to the head. The young practitioner must not hastily conclude that a patient is "only drunk," even if he be only confused, or if he swears or is violent, or if he lies on his back insensible, growling or swearing if disturbed. If he does, I am quite certain that he will have now and then bitterly to regret trusting to such circumstances. To have said that a patient was "only drunk" when a post-mortem examination shows a fatal lesion of the brain is very painful to all concerned. Besides, deep intoxication is itself a serious matter.

Injury.—We need not speak of cases where there is a clear history of very severe injury, because then the diagnosis is made for us. However, when the diagnosis has blindly rested on the fact that the patient has been in the way of injury, it is sometimes wrong. Prescott Hewett says (op. cit.), "There is no doubt that many a case reported as one of traumatic effusion of blood in the brain was simply a case of apoplexy."[1] In all cases of coma we search for bruises on the head and face. We examine the ears for discharge of blood, watery fluid, or even brain matter; the face for evidence of palsy of muscles supplied by the portio dura nerve—two things the frequent result of fracture of the base. We must be especially careful to note the condition of the conjunctivæ and eyelids, as effusion of blood here coming on *after* the injury, or after the patient was comatose, is evidence of fracture of the orbital plates. (By itself this is not, my colleague Mr. Hutchinson teaches, a serious symptom.) The absence of external signs of injury unfortunately does not negative serious and fatal injury to the brain. As before said, a slight fall may cause hæmorrhage into the arachnoid "cavity;" the heavy fall of a drunken man, or a fall in an epileptic fit. Even in cases of bruising and laceration of the cerebellum, the accident is not, Prescott Hewett says, always severe. In several cases the cerebellum was thus injured by the patient falling in the street when drunk.[2]

Even if we hear only that the patient has been in the way of injury some time before the symptoms set in, we must still consider the possibility of injury, as symptoms due to traumatic effusion of blood on the surface of the brain, especially if it be betwixt the dura mater and the bone, may come on, or at least develop largely, especially by a convulsion, sometimes hours or days after an apparently trifling injury.

If there be hemiplegia immediately after a fall, especially if the palsy does not follow a convulsion, non-traumatic hæmorrhage is most likely. Yet it is not quite certain, for there may be laceration of the hemisphere.

Opium poisoning.—In both poisoning by opium and *large* hæmorrhage, especially into the pons Varolii, there may be minute contraction of the pupils, universal powerlessness, and deep coma. "Contraction of the pupils is the most constant of all the effects of opium."[1] Hence there are on record cases of hæmorrhage into this part of the nervous system, mistaken for and treated as cases of opium poisoning. Unfortunately, there is not always minute contraction of the pupils in effusion into the pons, nor are they always contracted in opium poisoning. And in either condition, contracted pupils may dilate shortly before death[2]—"full active dilatation, which is uniformly observable when death (from opium) is imminent."[3]

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[1] Holmes' Surgery, vol. ii. p. 265.

[2] Op. cit. p. 312.

When we learn that the symptoms set in when the patient was with his friends, we must bear in mind that he may have taken the poison half an hour or even an hour before. We may detect the odour of opium in his breath. If there be a convulsion at the outset or soon after—cases of children are not here spoken of—we may almost certainly decide that there is not poisoning. My friend Dr. William Proctor, of York, however, has supplied me with notes of a case of rapid death of a woman in convulsion, after taking six grains of morphia. Caspar[4] says, "There are fits of spasms extending even to general convulsions." Scoresby Jackson[5] says that occasionally convulsions precede death. But these accounts of the symptoms refer to cases of children as well as of adults. I think we may say that in an adult, a convulsion—a severe convulsion at least, and certainly if it markedly affects but one side of the body—especially at the beginning, or soon after the beginning of the attack, nearly always negatives opium poisoning.[6] If we hear that the onset of the symptoms was very gradual—there being no albuminuria—we think that the patient was poisoned. Coma from effusion of blood into the pons Varolii will, it is true (see p. 553), come on deliberately, but not so deliberately as opium poisoning. In hæmorrhage the symptoms usually develop in a few minutes, or there is a sudden development of coma after slight symptoms. Soon after the poison has been taken the patient may be roused to give his name, but later he is in a state of as profound insensibility as clot ever produces. Moreover, the test is of little, if any, diagnostic value in cases of coma of any kind. If, however, we have no history, suppose the patient is found comatose in bed (we are supposing there are no local symptoms—such as palsy of the sixth nerve, turning of the two eyes or of the head to one side—that there is no convulsion), *we cannot make a diagnosis.* If the patient be a young adult, poisoning is probable; if past forty, apoplexy is more likely;

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[1] John Harley, The Old Vegetable Narcotics, p. 137.

[2] See Anstie, Stimulants and Narcotics.

[3] John Harley, op. cit. p. 138.

[4] Forensic Medicine, Syd. Soc. Translation, by Dr. Balfour, vol. ii. p. 63.

[5] Materia Medica, p. 330.

[6] It is right, however, to state that Tardieu describes one rare form of opium-poisoning which is not, so far as I can judge, to be distinguished from a case of large and rapid intracranial hæmorrhage. "Dans la forme foudroyante l'ingestion du poison est presque immédiatement suivie d'un sommeil comateux que rien ne peut vaincre ; la respiration est stertreuse et de cet état de narcotisme profond individus empoisonnés passent sans transition à la mort dans l'espace de trois quarts d'heure à une ou deux heures. Rarement celle-ci est precédée de quelques mouvements convulsifs. Une remarque est pourtant à faire dans cette forme, c'est que les pupilles sont constamment dilatées."

and I know of nothing in the pulse, in the respiration, or in the condition of the skin, which is of certain diagnostic value. An extremes lowness of the pulse, thirty or forty in a minute for a long time—say an hour or more—is said to favour the diagnosis of poisoning. But the pulse is sometimes rapid in opium-poisoning.

If the patient were dead when we were consulted, we should think he had not died of opium-poisoning if death occurred in less than six hours.[1] Poisoning by opium proves fatal in from six to twelve hours (Taylor). Effusions of blood into the pons, extensive enough to cause deep coma, will kill at varying times, from a quarter of an hour, which is rare, to twelve hours or more. However, we often have exceptional cases. Dr. William Proctor, of York, has recorded a case in which an ounce of laudanum killed a woman fifty years of age in less than two hours.

We have next to exclude epilepsy (see arts. Epilepsy and Convulsion).

Uræmia.—We now suppose that we find albumen in the urine. We have many times insisted on the fact that patients who are prone to Cerebral Hæmorrhage have frequently chronic renal disease. We cannot therefore logically attach much diagnostic importance to the mere presence of albumen in the urine. Practically it is not of value when the patient is past middle age, for his coma may be due either to uræmia or to Cerebral Hæmorrhage; and this is so whether the illness begins by convulsion or not. However it begins, we are sure there is not uræmia *only*, if there be hemiplegia, for then, if the patient be past middle age, we are practically certain that there is clot. But we are now supposing there is no discoverable paralysis. There are two chief ways in which uræmic coma comes on, without convulsion and then usually slowly, or rapidly and with convulsion.

If the patient known to be the subject of chronic Bright's disease, gradually becomes languid, and stupid, and as it were sleeps into coma, we may fairly diagnose uræmia. If the coma comes on suddenly, the diagnosis of clot is more likely; and if the coma be very deep, and the patient never moves nor can be roused to move any of his limbs, the clot is probably in the pons, especially if the pupils be minutely contracted. If, however, there is no history, we cannot tell when the coma is deep. I have known a patient found comatose in the street, from whose symptoms it would have been impossible to make a diagnosis betwixt clot in the pons (this was found *post mortem*), uræmia, and poisoning by opium.

Further, uræmia may begin suddenly, in the midst of seeming good health, by convulsion; but so may Cerebral Hæmorrhage (hemisphere or pons); and, to make the matter more difficult, these are the cases of Cerebral Hæmorrhage in which we often cannot make out any hemiplegia. If the convulsion were strictly limited to one side—most convulsions affect one side a little sooner and a little more than the other—I should for my part feel certain that there was not uræmia only, although of course we could not under these circumstances say there was Cerebral Hæmorrhage. (See art. Convulsion, p. 288.)

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[1] See, however, Tardieu, quoted in preceding footnote.

Serous Apoplexy.—This term is rarely used nowadays. Most cases so called were doubtless cases of uræmia. Yet we occasionally hear of cases of death by Apoplexy ascribed to "effusion of serum on the brain." If there be Bright's disease and, inferentially, uræmia, this may not be an altogether inaccurate description; for Traube considers that uræmic symptoms are directly dependent on œdema of the brain. These are doubtless, when there is no renal disease, cases of what are here called "Simple Apoplexy." By this term is to be understood cases of Apoplexy in which no lesion is discovered; that is to say, no lesion which we can suppose to have been the cause of so dramatic a mode of dying. One reason why these cases are called serous is probably that there is not unfrequently found at the autopsy a large quantity of serum in the meshes of the pia mater. But this is really a chronic state of things, and so far from the fluid exercising pressure, it has simply been "effused" very gradually to take up the room vacated by wasting of the brain. This is seen strikingly in cases in which there is wasting of but one cerebral hemisphere; here the serum is "effused" on one side only.

Simple Apoplexy.—Now, supposing we have excluded drunkenness, injury, epilepsy, and uræmia, we have still to determine whether the case be not one which for want of better knowledge we can only name from its negative post-mortem appearances, Simple Apoplexy. We have already (art. Convulsion, p. 291), when speaking of patients dying after attacks of convulsion, stated that in some we find marked changes *post mortem*, and in others we discover nothing abnormal in any part of the body. But patients pass into deep coma when no convulsions have been observed, or after apparent recovery from a convulsive seizure. A patient, sometimes even a young man, quickly becomes apoplectic and dies in a few hours, and in the whole body we find nothing abnormal which can reasonably be supposed to have been the cause of the symptoms. This class of cases is well recognised. Dr. Todd[1] says, speaking both of delirium and coma, that "both these formidable states may take place in a brain which shall reveal on the minutest scrutiny no appreciable aberration from the natural standard." Dr. Wilks says in his Lectures on Pathology: "Occasionally you may be called to a case where the patient is insensible or suffering from apoplexy, and on examining the brain you find nothing. During the last two years I have seen two cases where the post-mortem revealed nothing." It seems certain that these patients die from the brain. At all events they die in the same way as patients do who die in coma from Cerebral Hæmorrhage, and in such cases during life Cerebral Hæmorrhage is frequently diagnosed. The post-mortem appearances of the heart and lungs are such as those we find in patients who have died with large cerebral hæmorrhage.

I freely confess that I know of no rules by which to distinguish simple from sanguineous Apoplexy, or other forms of coma. We cannot rely on the kind of pulse, nor on the temperature, nor on the state of the pupils, nor on stertor. I have, in short, nothing to say of diagnosis here.

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[1] Nervous Diseases, chap. viii.

I have observed that some medical men seem, if I may use such an expression, to be disappointed in not finding in the head of a patient who has died in an apoplectic manner, anything which can be supposed to have given rise to his symptoms. In these cases the suspicion of poisoning will occur. Indeed, this possibility ought to be carefully considered. Yet this part of the question is legal rather than medical, and at an inquest we can assure the coroner—who, if he be a medical coroner, requires no strong assurance on that matter—that the profession recognises such cases as cases of natural death.

Some of these cases are put down to congestion of the brain. But this conclusion is often drawn from the distension of the cerebral veins, which is a very common appearance in patients who have died rapidly from any cause; and in all cases, even in cases of death from hæmorrhage, we find fulness of the veins in the occipital region.

For the diagnosis of Apoplexy from *Congestion of the Brain* and from *Sunstroke* see those articles.

Aneurism of the larger cerebral vessels. (See arts. Adventitious Products and Convulsion, p. 288.)—Cerebral aneurism has been incidentally considered in several parts of this article.

Hæmorrhage from Tumours.—Occasionally fatal hæmorrhage occurs from cerebral tumours. We can only make the diagnosis from the evidence supplied by a history of tumour of the brain (see art. Adventitious Products), and if there be no history we cannot make a diagnosis. If, however, in a young patient we discover double optic neuritis, we should suspect tumour.

Occasionally apoplectic symptoms come on suddenly from *Abscess of the Brain* (see art. Abscess of the Brain). We can only make the diagnosis from such facts as the history of a blow, presence of "puffy" tumour, disease of the ear, &c., and when these facts are not forthcoming we cannot make a diagnosis.

Prognosis.—Here we speak of Cerebral Hæmorrhage only. It is again to be insisted on that Cerebral Hæmorrhage is not a constant quantity; the clot varies in size, in suddenness of effusion, in position, and there are differences in the ages of the patients attacked, and in their constitutional condition. Obviously then we can only speak very generally on prognosis, and what would properly come under this head has been already in chief part considered. Thus, under Etiology, we pointed out that in many cases the constitutional condition of the patients who suffer Cerebral Hæmorrhage is one of wide-spread degeneration. If therefore the symptoms which we attribute to Cerebral Hæmorrhage be in themselves trifling and transitory (see Premonitory Symptoms, pp. 549–50), they are of very evil omen if the patient be past middle age, and if there be hypertrophy of the heart, degenerated arteries, and chronic renal disease. But here the evil omen is as to the future. We speak next of cases of larger hæmorrhage, and of prognosis as to recovery from hemiplegia, or apoplexy, or, as is usually the case, from both. Of course the graver the lesion, the worse the prognosis. We estimate its gravity by the degree of the paralysis and by the degree of the apoplectic condition. Under Localization, p. 537, it was pointed out that the more *complete* the paralysis the graver the lesion. Thus if the patient has, besides palsy of the face, arm, and leg, lateral deviation of both eyes and of the head, the worse the prognosis; if he escape with his life, palsy of the face, arm, and leg will almost certainly remain. If the palsy be incomplete, the prognosis is less grave, both as to life and recovery from paralysis. But we cannot judge by the paralytic symptoms alone. The degree of the apoplectic condition is to be considered also, although it is usually greater in degree the more complete the palsy. The less, and the more transient, the loss of consciousness, the better the prognosis; the deeper the loss of consciousness, the worse the prognosis. The prognosis is very grave indeed if the patient, after being simply hemiplegic, becomes suddenly profoundly unconscious and universally powerless, and it is graver still if the change sets in by convulsion, for this mode of ingravescence points to rupture into the lateral ventricle. The other symptoms of the apoplectic condition are to be considered. The more the pulse, respiration, and temperature are implicated—either depressed in the first stage or raised in the second—the graver the prognosis. In other words, the more the automatic processes are involved, the worse the prognosis. We have seen (p. 537), that along with degrees of loss of consciousness there are in different cases all degrees of range of palsy, palsy of the most voluntary parts (face, arm, and leg), palsy of these and of more automatic parts (deviation of the eyeballs and head, &c.), and even palsy of the *most* automatic parts as evidenced by stertor and depression of pulse, respiration, and temperature.

When the patient has come round from the apoplectic condition, his condition varies. Since there are all degrees of gravity of the lesion, there are all degrees of the conditions left when the apoplectic symptoms have passed off. The deeper and the more continued the apoplexy has been, the worse the after condition of the patient is likely to be. He may be, especially when speechless, in a state of complete imbecility, lying in bed, taking no intelligent notice of what goes on, and passing urine and fæces in bed. Although frequently he eats voraciously, he gets gradually thinner and often dies in a few weeks or months. In other and less severe cases, there is great defect of memory, especially for recent events, and great emotional instability; the patient is easily made to laugh or to shed tears, though he does not laugh with any healthy ring, and his crying is a blubbering, very painful to witness. There is also great irritability of temper and often a heedless selfishness; the patient's disposition, his friends tell us, is quite changed, his mental field is narrowed: he seems to care much for his own immediate wants, and cares little about his family or business concerns. In other cases there is little more than paralysis, although the patient's mental condition is not so good as before.

The palsy often diminishes, and improvement follows a certain order. The more automatic parts recover first. Thus the lateral deviation of the two eyes and the head usually passes away in a few hours or days. The leg is the next part to recover, although it rarely recovers completely after severe apoplexy, and the lingual and facial palsy diminish or pass away altogether. When rigidity of the limbs comes on, we fear no further improvement will follow. We may find the patient speechless[1] (aphasic), on recovery from the apoplectic condition, and he usually remains so if the apoplexy has been deep and continued.

If there has been no loss of consciousness, or only transient loss of memory, the patient has a good chance of recovering altogether from the paralysis and the affection of speech. But as we have seen (p. 553), it is not easy to be sure that hemiplegia without loss of consciousness is owing to clot. Most cases of this kind are owing to softening from embolism or thrombosis. Recovery from hemiplegia will occur from any kind of lesion if it be a small one. We can only judge by the early beginning of the recovery. If the patient begins to move the arm next day, he is likely to get well altogether. We cannot infer so much from early recovery of the leg, as this is very often not completely paralysed at the outset, and we know that it frequently recovers when the arm remains much paralysed.

TREATMENT.—The recovery of the patient, it is most probable, depends altogether on the quantity and seat of the hæmorrhage. If the ventricles be opened, if there be a large clot in the pons, the patient will die. But as in many cases we cannot be absolutely sure that there is any hæmorrhage we must treat the apoplexy (see arts. Softening and Renal Diseases). We must particularly bear in mind that if the cause of the apoplexy be alcoholic poisoning, recovery usually follows, even in very severe cases. When in doubt we should use the stomach pump.

There is unfortunately little to be done, in cases of large Cerebral Hæmorrhage, and the chief thing is simply to keep the patient quiet, especially when we see him soon after the attack. Rousing him may lead to such increase in the size of a clot in the brain that it breaks into the ventricle.

Bleeding used to be almost a routine practice. In this country it has fallen into disuse. Although I have observed very many cases of Cerebral Hæmorrhage, not only in my own practice but in that of others, I have seen but one patient bled for it. I quote, however, part of what Niemeyer says on this point. It will be observed how carefully he tries to distinguish the cases in which bleeding is admissible from those in which it is hurtful:—" If the impulse of the heart be strong, and its sound loud; if the pulse be regular, and no signs of commencing œdema of the lungs exist, we should bleed without delay. Local bleeding by leeches, behind the ears, or to the temples, or by cups to the back of the neck, cannot replace general bleeding, but may be used as adjuvants. If, on the contrary, the heart's impulse is weak, the pulse irregular, and rattling in the trachea has already begun, we may be almost certain that bleeding would only do harm, since the action of the heart, which is already weakened, would be still more impaired, and the amount of arterial blood going to the brain would thus be still more decreased. When the latter state occurs, the symptomatic indications require just the contrary treatment, in spite of the original disease being the same, and being due to the same causes. We must strive with all our skill, by the use of stimulants, to prevent paralysis of the heart. If we cannot give wine, ether, musk, &c., internally, we should apply large sinapisms to the chest and calves of the legs, rub the skin vigorously, sprinkle the breast with cold water, or drop melted sealing-wax on it."

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[1] See art. Softening for an account of Aphasia.

It must be difficult to select the right time as well as the right case, as the pulse, respiration, and temperature are in very different conditions at different stages in the same patient (see p. 547). In the first stage the pulse may be very slow, and the temperature greatly reduced. We should rather give stimulants than bleed in this condition, but I think it is better not to do this, unless the pulse be very feeble, and the temperature much reduced; we may also apply mustard plasters to the calves. When the pulse and respiration become very quick, when there is evidence of engorgement of the lungs—as shown by the loud rattles we hear from œdema of the lungs—we might, from theoretical consideration, suppose that bleeding would be of service by relieving the venous system, which is evidently overcharged. But at this time the pulse is really feeble, and occasionally it is irregular.

It is, I think, good practice to give croton oil in either stage, unless the alteration of pulse, respiration, and temperature be extreme. In the second stage—the stage of reaction, and when the clot is producing a local encephalitis—it is well to apply cold to the head. Blisters relieve the severe headache in the cases of cerebral tumour (no doubt often one of the symptoms of a local encephalitis), and it is possible that blisters to the back of the neck, and behind the ears, are of service when the patient is recovering from the apoplexy, and has pains in the head.

Let us now suppose that the apoplexy is past, or that there has been no unconsciousness, or a very temporary confusion when a hemiplegia, indicating effusion of blood, came on. The more vividly we realize the fact, that a mass of blood is lying abroad in softened and torn nervous tissue, the less confident do we feel in our power to interfere for the patient's good. The feeling of helplessness is greatest, when we are looking at a clot lying in nervous tissue, *e.g.* in the retina. There is, to my knowledge, no treatment for effusion of blood in nervous tissue.

There are no drugs which assist in the absorption of the clot. However, it is quite certain that some patients recover satisfactorily from hemiplegia, the result of Cerebral Hæmorrhage. But recovery from hemiplegia will follow when damage to the motor tract remains. From not recognising this fact, erroneous conclusions may be drawn as to the effects of remedies. We must particularly bear in mind that anæsthesia disappears or diminishes quickly when no drugs are given, and also that there is a natural order of recovery, as stated under Prognosis (p. 565), which probably is not interfered with by treatment.

We have still, as in many other diseases, to improve the general health. This is, however, not unfrequently, rather general disease, and the local lesion—let us say epistaxis, paralysis for a few days, or a day's thickness of speech—is sometimes a small matter in comparison with the state of the system of the patient who comes to us for such slight symptoms. The proper care of a patient who has a clot of blood in his brain, and who is liable to have further effusions, consists in attending to his diet, excretions, sleeping and exercise. Care in diet is especially important. In this connexion we may quote what Niemeyer says (op. cit. vol. i. p. 314), under the head "hypertrophy of the heart." We have seen that in most cases of Cerebral Hæmorrhage, there is cardiac hypertrophy, and in many cases the nervous symptoms, when the clot is small, may be almost unimportant in comparison with the unsound state of the system:—"Such patients must beware of immoderate eating and drinking, in order to avoid the plethora which, although but transient, always follows upon a free use of food or drink. How often does the long-threatening apoplexy set in in the midst of the plethora which has developed after a long and hearty meal! . . . . In this connexion I may mention an act of folly which I have often seen practised by tavern-keepers and itinerant wine-dealers. The latter often suppose that, by a free use of water, they can counteract the pernicious influences to which they expose themselves, although it is evident that the plethora arising after a full meal would only be increased by an immoderate addition of fluid. Besides this, however, the patient must avoid all the causes which, independently of plethora, stimulate the action of the heart, and further distend the already overcharged arteries. Under this head come the use of stimulating drinks, mental excitement, and immoderate bodily exertion. Hot water must be included in this class, and there is no wonder that the use of the Karlsbad Sprudel should make victims every year who die of apoplexy."

ABSCESS OF THE BRAIN.

By William W. Gull, M.D. F.R.S., and Henry G. Sutton, M.B.

Abscess of the Brain is comparatively a rare disease, and it falls to the lot of no man to see a great many cases. We have collected seventy-six cases in all from various sources, and the details in this paper are based upon these records. Many of the cases have not before been published. We have arranged the different parts of this subject in the following order:—A description of the various conditions that are known to give rise to cerebral abscess, the morbid anatomy, the symptoms, pathology, diagnosis, and treatment.

Suppurative inflammation of the brain may be caused by injury to the head, especially where the skull is fractured and the brain contused. Mr. Prescott Hewett says: "All traumatic inflammation of the brain substance may end in suppuration and abscess."

Cerebral abscess may follow a penetrating wound of the brain substance, by a knife, by a splinter of wood, or by some sharp instrument being forced through the skull (Case 74).

Abscess of the brain may follow a fracture of the skull where there is no displacement of the bone; acute suppurative inflammation of the membranes and brain substance being set up by the injury (Case 1). In many cases, caused by fracture of the skull, the abscess in the brain is seated immediately under the injured bone, and close to the surface of the hemisphere. In others the abscess is not seated near the surface; for instance, a person may receive a fracture of the skull, symptoms of compression may set in, and the skull may be, in consequence, trephined; the portions of depressed bone may be removed, and the patient go out of the hospital apparently well. But after a few weeks or months, cerebral symptoms may again appear, and the patient may die; and the autopsy reveal an encysted abscess embedded in the substance of the brain, and seated at some distance from the surface (Case 2).

Cerebral abscess may follow an injury to the skull, where there is no fracture of the latter, and with (Cases 15 and 53), or even without a scalp wound. In such cases the injury excites inflammation and suppuration of the diplöe of the bone, and the suppuration extends and involves the brain.

Cerebral abscess may follow contusion, or, as it is sometimes expressed, concussion of the brain, without there being any fracture or other discoverable injury to the skull. Mr. Prescott Hewett says that he has seen two cases of this kind, and the abscesses were large.[1]

This is a very important class of cases, for it probably embraces not a few of the so-called idiopathic abscesses of the brain.

In two of our cases, abscess was found in the brain, though in neither was there any evidence to show that the skull had been fractured or otherwise injured. With both patients the symptoms followed directly after the injury; one had a fit on the same day as the accident, and the other suffered from almost constant pain in the head for a fortnight after the accident, and was otherwise generally indisposed. The abscesses were encysted in both instances, and, during the time they were forming, there were symptoms indicative of cerebral disease, although, in the second case, the symptoms were, for a while, obscure. One patient died seven weeks, and the other three months after the accident.

Cases might be given to show that abscess may follow injury to the head, without any fracture or other discoverable injury to the skull (Cases 7, 13, 20, 35, 39, 43); and the abscess may remain latent for months or even longer.

One of the commonest causes of cerebral abscess is disease of the internal ear. The clinical history of this class of cases is usually as follows: the patient has a discharge from the ear for some time—for months—and, in many cases, for years; the discharge being continuous or intermittent. It is common to hear it said that the discharge began in childhood, after an attack of measles, scarlatina, or small-pox; and since has returned, more or less. With the discharge there is often deafness and pain in the ear, but more often the patient makes no complaint of either. In some cases, the discharge is very offensive, and has been so for some time past. The extension of the disease to the brain is often very insidious. There may be no indications that the brain has become seriously involved until acute symptoms set in a few days before death. Very often the first sign is a great increase of the pain in the ear. The pain is often very severe, and comes on in paroxysms, so violent in some cases, that the sufferer screams with it. Occasionally the acute mischief in the brain is ushered in with rigors; at other times with nausea and vomiting.

Sometimes an epileptiform convulsion ushers in the acute symptoms, and a few days after this the convulsion is repeated, and followed by hemiplegia.

The accession of acute symptoms appears, in many cases, to correspond with the commencement of acute inflammatory softening, either primarily in healthy brain, or secondarily around an old abscess. Then the skin becomes hot, the pulse quick, tongue dry and parched; great prostration, drowsiness, and stupor set in,—such symptoms as resemble continued fever, and have been mistaken for it in some cases. The discharge from the ear varies very much during the acute symptoms. It is common for it to subside, or even entirely to disappear.

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[1] Holmes' Surgery, vol. ii. p. 185.

Chronic changes, dependent upon diseases of the internal ear, may be insidiously going on in the brain substance, without there being any symptoms of cerebral disease.

Mr. Toynbee was of opinion that the inflammation extends to the brain, from the pus not escaping from the cavity of the tympanum externally. He says: "So long as there is a free exit for the discharge, I believe the disease rarely extends to the brain."[1] He also remarked: "In all fatal cases the discharge has been deprived of a free egress." Mr. Toynbee further stated, in cases where the disease attacks the mastoid cells in early life, the cerebrum is the part of the brain which is most likely to suffer, while in later periods of life the cerebellum is the part most generally affected. Long experience has clearly shown that, when disease of the internal ear has gone on for a long time, the temporal bone is very liable to become diseased. When the patient dies with cerebral symptoms, it is common to find caries of the petrous, or mastoid, portion of the temporal bone. It is also common to find suppurative inflammation of the dura mater covering the diseased bone, with or without sloughing of that membrane. There is, in some cases, no direct extension of the disease from the bone to the contiguous parts. In such cases the bone, membranes, and surface of the brain are healthy. A portion of healthy brain may lie between the abscess and the bone. The diseased action is considered to extend by a vein. It is rare to find abscess of the brain following acute disease of the ear; but one case is alluded to by Mr. Toynbee.

In cases of chronic disease of the ear, the causes of the acute brain mischief are various. A blow on the head, violent exercise, or other depressing influence; also cold air, or some irritating application, is sufficient to engraft acute changes upon the chronic disease.

Cerebral abscess may be associated with, and apparently dependent upon, chronic disease in the lungs; but in two of our cases the morbid appearances were such as to indicate acute changes in the lungs, extending, however, over several weeks (Cases 9 and 57).

In a case that occurred in St. Bartholomew's Hospital, the lung presented the appearance of acute pneumonia in the third stage; but the symptoms indicated that the disease had been going on about two months and ten days (Case 9).

In all the other cases which have come under our notice, the morbid changes in the chest had evidently been going on several months and even years (Case 38). In one, there was a large suppurating chronic empyema (Cases 10 and 11). In another, there was a large cavity at the apex of the right lung, which was firmly adherent to the chest walls by a thick layer of indurated tissue. Another patient had had flattening and general contraction of the left chest for years, signs of dilated bronchial tubes, and of disease in the left lung (Cases 36 and 38).

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[1] *Vide* Diseases of the Ear, by Mr. Toynbee, p. 303.

Suppuration in any part of the body may give rise to secondary abscess in the brain. In one of our cases there was an abscess in the sheath of the left rectus abdominis muscle, and several abscesses without cyst in the brain (Case 32). In this case it is instructive to notice that the lungs, the common seat of pyæmic abscesses, did not contain any abscesses, nor were there any in the liver or spleen. In another case there were pyæmic abscesses in the brain (Case 33), apparently the result of chronic suppuration of a mesenteric gland and co-existing recent abscesses in the spleen and kidney. In a case of acute necrosis of the tibia (Case 4), which occurred in St. Thomas's Hospital, there were numerous abscesses in the brain, and pyæmic abscess in the lungs, liver, and spleen. In a case given by Dr. Bright, a whitlow was the source of general pyæmia and abscess of the brain. In another case, referred to by Lebert, the drawing of a tooth was followed by inflammation of the upper part of that face and cerebral abscess. Dysentery was the cause in one instance (Case 25); abscess near the uterus (Case 8); suppuration in the Fallopian tube (Case 75); carcinoma of the face (Case 50); abscess in the liver (Case 51), and the phagedænic ulceration, following amputation of the breast (Case 56), were the causes in other cases. Dr. Ogle relates a case of secondary purulent deposit in the brain, apparently the result of ulceration of the cæcal appendage. There is also another recorded case following amputation of the forearm.[1]

In chronic disease of the bones of the nose, and in cases of syphilitic disease of the bones of the skull, there is a liability to cerebral abscess (Case 41).

Morbid Anatomy.—An abscess may form in any part of the brain. Usually it forms in the white substance, and when in the grey it is formed by extension from the white. The middle cerebral lobes are the most frequent seats of abscess. One hemisphere is as frequently attacked as the other. Of 80 cases, abscess was situated in the left hemisphere in 23, and in the right in 29. Practically, therefore, one hemisphere would appear to be as liable to be attacked as the other. In 12 cases abscess was situated in the middle lobe, but it is not stated in which hemisphere. The middle lobes were the seat of abscess in 23 out of 74 instances. Abscess was found in the cerebellum in 13 cases, in the pons Varolii twice, in the corpus striatum twice, in the optic thalamus twice. Abercrombie mentions an instance of abscess in the medulla oblongata. In several of the 74 cases the abscesses were multiple, and found in more than one part of the brain. The appearance of the abscess varies according to its duration. If it have been recently formed, the pus is not enclosed in a cyst, but directly surrounded by ragged suppurating brain tissue, and there is not a trace of lining membrane to the cavity. If the abscess have been formed some time, the pus is enclosed in a cyst of variable thickness. In very old abscesses the cyst wall has been found a quarter of an inch, or more, in thickness. When the abscess is a few weeks old, the cyst wall is usually a line or two in thickness. The wall of the cyst is formed of fibro-cellular elements, and, in some cases, well-formed spindle-shaped fibres are seen; in others the fibro-cellular tissue has undergone granular degeneration, and the fibre cells are very indistinct. The cyst, when of old date, may be divided into three parts—an outer layer, which is made up of loose, fine, fibrous tissue; a middle layer, which is firmer and more coarsely fibrous than the outer; and the inner surface of the cyst is formed by a smooth, pyogenic membrane, in which some small irregular dilated veins may be seen running in different directions.

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[1] From analogy we should expect that an hydatid tumour, or a so-called strumous deposit in the brain, would cause abscess. We have, however, no record of such a case. Abscess is also said to have occurred when the carotid artery was tied. Probably it was softening of the brain, and not abscess.

In abscesses of recent formation, the pus is generally of a greenish hue, and may, or may not, have a disagreeable smell. In old abscesses, the pus is green, fœtid, mucoid, and is decidedly alkaline. The pus removed from old abscesses, when placed under the microscope, shows few or no well-developed pus corpuscles; there is a large quantity of granular fat and granular matter without any nuclei.

There may be several encysted abscesses in the brain. In one of our cases there were no less than four; in another a large encysted abscess in each hemisphere.

The condition of the brain substance immediately around the abscess may vary very much; it has commonly undergone a process of softening. Rokitansky, speaking of recent abscess, says, round the abscess the brain substance is in a state of inflammation, producing red softening, yellow softening, and in more distant parts œdema of the brain tissue.

When a large abscess is situated in one of the hemispheres, the brain is often altered in shape; the convolutions being packed together and flattened; the hemisphere bulged at the side, and if the abscess be very large, the hemisphere containing it may feel more like a bag of pulpy thick fluid than solid brain substance. Collections of pus, in the hemispheres, tend to make their way towards, and discharge themselves into, the lateral ventricles, or on the surface of the brain. Pus, like blood, may fill one lateral ventricle only, or escape into the ventricle on the opposite side. In abscesses, as in very vascular, soft, gliomatous tumours of the brain, hæmorrhagic effusions are occasionally met with, and a coagulum of blood may be seen surrounded by pus.[1]

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[1] See Guy's Hospital Reports, vol. iii. 3d Series, Case No. 6, p. 291.

We have already stated that several abscesses may exist together in the brain; this is common when a patient has died of pyæmic cerebral abscess. In such cases every part of the brain may be studded with minute collections of pus; they may be found in the cerebrum, in the cerebellum, in the optic thalamus, in the corpus striatum, and pons Varolii. The size of these abscesses may vary from a pin's head to a hazel-nut, or even larger. They are usually situated near the surface of the brain. The cerebral substance around these pyæmic abscesses may be softened, at other times it is firm and comparatively healthy. When abscess of the brain is dependent upon disease of the internal ear, the morbid appearances are much as follows: the dura mater, situated over the diseased petrous or mastoid portion of the temporal bone, is often found highly congested, softened, and ulcerated; or of a dirty green colour, and evidently sloughing, and the bone laid bare. In other cases the dura mater is simply thickened and covered with purulent lymph, and betwixt the dura mater and the bone there is often a collection of pus. The lateral sinuses are frequently involved and plugged, especially when there is disease of the mastoid cells; the sinus is often seen enveloped in pus and purulent lymph. The suppurative inflammation may extend along the internal jugular vein, and set up suppurative pleuritis and abscess in the lung.

In abscess of the brain due to disease of the ear, there is, in the majority of cases, caries of the temporal bone; the latter is seen of a dark colour, with an irregular roughened surface. The abscess in the brain may have direct communication with the diseased bone, and the contents of the abscess make their way through the ulcerated openings in the dura and bone into the tympanum, and then escape through the perforated membrana tympani into the external meatus, thus constituting what has been termed "otorrhœa cerebralis." A similar communication and escape of the pus is said to have occurred in cases of abscess in the brain caused by diseased ethmoid bone. At other times there is no such direct communication, for there is a layer of brain substance separating the abscess from the membrane of the brain. This layer is often softened, of an ash grey, or yellowish appearance, and looking as if the pus were about to burst and discharge itself on the surface of the brain.

In some cases of abscess dependent on disease of the internal ear, there is no caries of the bone, as we have already mentioned, the membranes may be healthy, and the abscess may be situated at a distance greater or less from the surface of the brain.

SYMPTOMS.—In 73 cases of abscess of the brain, the symptoms were as follows:—Pain in the head in 39 cases; epileptiform seizures in 38; coma in 30; heaviness, stupor, and drowsiness in 30; paralysis in 24; rigors in 17; pyrexia in 13; delirium in 13; vomiting in 12; incontinence of urine, or of fæces, or both, in 15; vertigo in 8; disordered sensibility, not including pain in the head, in 6; defective articulation in 4; defective sight in 3; an apoplectic attack in 1.

That some of the symptoms may have existed in greater proportion, we should be prepared to expect, especially such symptoms as vertigo, pyrexia, emaciation, and probably in a greater number of cases, defect of sight would have been discovered had the eye been tested. The symptoms, therefore, that are most frequently observed in cases of abscess in the brain are pain in the head, epileptiform attacks, paralysis, coma, heaviness, drowsiness, stupor, rigors, pyrexia, delirium, vomiting, and incontinence of urine and fæces. In a few cases defective articulation was met with. The records show that the intellect was very little affected. Paralysis was observed in 24, that is in about one-third, whereas in Lebert's cases it was observed in about one-half. He included, however, not only local paralysis, but also general loss of muscular power, whereas we have confined the term to local paralysis only, such as loss of power on one side of the body, of one arm or leg, one side of the face, or some other part.

The first symptom, in many cases, is pain in the head; it may be the only indication of cerebral disease present for months. The pain is often very agonizing.[1]

An intense neuralgic pain situated over one spot is occasionally the first symptom; sometimes the pain is seated almost immediately over the region of the abscess. A boy having an abscess in the anterior lobe of the right hemisphere, complained of almost constant burning pain over the front and right side of the head, but this localization of pain over the seat of the abscess is by no means constant. In some cases the pain is very remote. In one patient there was an abscess in the cerebellum, and the pain was felt in the forehead; in another there was an abscess in the right middle cerebral lobe, and the pain was referred to the left side of the head.

The pain often comes on in paroxysms; in other cases it is continuous, remittent, or intermittent. It is not present in all cases of cerebral abscess, as the statistics of our 76 cases show. It is very commonly associated with pain in the ear, when the abscess is due to disease of the auditory apparatus.

Instead of pain preceding, it may follow the convulsive attacks. Cases of this kind are by no means few.

Occasionally the first indication of cerebral mischief is a sudden and unexpected epileptiform seizure. The epileptiform seizures are occasionally the most prominent symptoms from the time of seizure to the patient's death. The epileptic attacks do not necessarily come on every day; occasionally some days elapse between the seizures.

After each convulsion the side affected is often left weak, and this increases until there is complete hemiplegia. The convulsive movements are sometimes unattended with insensibility, and are confined to one extremity, especially the arm. This has been long noticed.

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[1] One patient lay in bed continuously holding his head with both his hands; another walked about with his hands pressed against one side of his head, crying out constantly, "Oh! my head; oh! my head." The pain is often so severe that the patients shriek from the agony they suffer. A patient, who was perfectly sensible, said he could not help screaming; and, although he tore and bit anybody or anything near him, he at the same time expressed contrition for what he was doing, and said the pain in his head was unbearable; it felt as if some one was knocking it with a hammer.

Abercrombie alludes to a case of Lallemand's, in which there was pain in the right side of the head and tremor of the left arm. This was followed by continued convulsions, flexion, and extension of the left arm, which after some days ended in palsy.

Instead of convulsive movements, the first indications of brain disease may be numbness and tingling in one extremity.

The symptoms in other cases of cerebral abscess are like those that are said to indicate cerebral softening. There is sudden loss of power on one side of the body without any loss of consciousness; the leg being less affected than the arm.

In several instances rigors were very prominent symptoms throughout the attack. A patient, suffering from suppuration, was noticed to be getting thinner and weaker; then he was seized with rigors, diarrhœa, a dry brown parched tongue, and a hot skin; he became comatose and died. Pyæmic abscesses were discovered in the brain.

In some cases of pyæmic abscesses, there are no special symptoms to show that organic disease is going on in the brain; but only the general indications of pyæmia. In others the accession of convulsive seizures, paralysis, or coma, indicates disease in the cerebral organ. Rigors were noticed in a few instances so severe, and returning with such regularity every day, that they closely resembled those of ague. One patient had headache, rigors, and vomiting, returning every day for five days, and then became unconscious. Rigors do not occur, in some instances, until after convulsive seizures have indicated cerebral mischief. Imperfect articulation, to a marked degree, was noticed in some cases, and in one there was loss of language.

With respect to the eye, Dr. Hughlings Jackson has mentioned to us that he has seen changes in the retina (optic neuritis ?) in a case of cerebral abscess. Dr. Jackson thinks such changes are common to several kinds of cerebral disease.

Mental disturbances were observed in some cases. Now and then, the only symptoms noticed were a heavy expression, a disinclination to speak, and indifference to surrounding objects. In some cases with disease of the ear, it was stated that the patients had attempted to commit suicide. One patient appeared to become hypochondriacal. Emaciation setting in rapidly was a marked symptom in several cases. Similar emaciation is seen in some cases of tumour of the brain; but is not so frequent as in abscess.

Patients suffering from cerebral abscess may have symptoms so closely resembling continued fever, that it is exceedingly difficult, if not impossible with any degree of certainty, to say whether it be a case of fever or of organic disease of the brain.

PATHOLOGY.—Cerebral abscess may be produced by direct injury, or by contre-coup, contusing or lacerating the nervous tissue, and setting up inflammation and suppuration. It may be produced by suppurative inflammation in some tissue in the neighbourhood of the brain which spreads to a contiguous part; namely, in the ear or nose, which extends, and invades the dura mater, pia mater, and brain substance. Or the diseased action may spread by continuity of structure, as along a vein, and thus to the brain. Disease of the ear, nose, or of other cranial bones, may give rise to cerebral abscess in this manner. Again, abscess may be produced where there is disease of the cranial bones, or some growth involving them, by the veins communicating with the diseased bone becoming plugged. The process of coagulation extends and invades the veins communicating with the sinuses of the dura mater. These become plugged, as also the veins of the pia mater and probably some branches entering the brain tissue also, and inflammation, terminating in suppuration, is thence set up in the brain. In other cases, minute coagula, or thromboses, are supposed to be detached and carried along by the circulation until they are arrested in the capillaries of the brain, and often of the lungs, kidneys, and other organs.

Pyæmic abscesses are occasionally found in the brain, and not in any other organ of the body. Besides the coagula, some of the elements of pus may be carried by the circulation to aid in, or be the means of, setting up suppuration in the parts where the thrombosis is arrested. In this way abscesses in the brain are probably caused by abscess or suppuration in the liver, lungs, bowels, or in other parts.

We next inquire if every form of cerebral inflammation, or encephalitis, no matter what its origin, be liable to end in suppuration and an abscess. It has been many times stated that such is the case; but it would appear that the inflammation must be set up by a special cause, and unless it be so, it does not end in suppuration and abscess. Suppuration may apparently be excited by local injury, or by the elements of pus or thrombosis; but experience shows that other forms of inflammation do not terminate in abscess. For instance, encephalitis and softening, the result of plugging of a cerebral artery, or encephalitis around a hæmorrhagic effusion, or around a gliomatous tumour or old cyst, shows no disposition to the formation of pus or abscess. The brain may soften, disintegrate, and a cyst may be formed, but there is no pus formed.

It is necessary, now, to ask if there be not good evidence to show that the brain may be the seat of suppurative inflammation and abscess without there being any cause to account for it? Is there not, in such cases, idiopathic inflammation which gives rise to idiopathic abscess? By idiopathic cerebral abscess, we suppose, is meant abscess which is not preceded or occasioned by injury or disease; its origin being unaccounted for. Lebert and others admit the occurrence of idiopathic cerebral abscess. Such cases are, however, in comparison with others, rare. It is beyond all doubt that a certain number of cases of cerebral abscess do occur in which no disease is discovered in any other part of the body, and there is no history of any recognised cause to account for the cerebral abscess.

Before, however, it be concluded that abscess has been formed idiopathically, it is necessary to remember that in the majority of cases there is a cause to account for the formation of such abscess, and that only in a very small minority have observers failed to find some admitted cause. In the face of such evidence, is there not good reason to think that, in this small minority of cases, the primary cause has been overlooked? And, when it is still further remembered that hours have been passed in searching for the primary disease or cause, and at last it has been found limited to a mesenteric gland, a gum-boil, or a whitlow—in fact the primary disease was so small, that it might have been very easily overlooked—it appears to us not difficult to understand how, even after very great care, the primary cause may have remained undiscovered. Bearing all this in mind, we recognise that in a few cases of cerebral abscess, the cause cannot be discovered; but even when the cause is undiscovered, we should not assume that the suppurative inflammation has commenced idiopathically in the brain.

Cerebral abscess proves fatal in many cases, not by a collection of pus in one or other part of the brain, but by extensive inflammatory softening around the abscess, involving vital parts of the brain; and it is from such softening that the abscess is enabled to make its way towards the ventricles or the surface of the brain. The softening around very old encysted abscess would appear not to be set up by pyogenic changes going on in its lining membrane, for there is not a large quantity of well-formed pus corpuscles in old encysted abscesses to show that such active changes have been going on in this membrane.

The softening would rather appear to be due to some circumstance interfering with the nutrition of the parts outside of the abscess, but in its neighbourhood. The nutrition of such parts, owing to the presence of a foreign body, being very feeble, it is easy to understand how a blow on the head or a debilitated or cachectic state of the system may be sufficient to excite such feebly nourished parts to take on acute inflammatory softening.

Has abscess in the brain any tendency to spontaneous cure? Lebert thinks not; and when we remember that there is no well-established case on record, showing that an abscess has been spontaneously cured, we readily admit that the evidence very strongly favours the belief that cerebral abscesses do not tend to a spontaneous cure. It is, however, necessary to remember that the brain is a very vital organ, severely taxed in our every-day labours, and, if not sound, its functions, which are essential to life, may be brought to a stop. When there is an abscess in the brain, the organ being unsound, its functions are very liable to be perverted, and death follows; whereas, if the abscess were seated in an organ less essential to life, any perversion of its functional activity would not be attended with fatal results, and thus time would be gained for the abscess to pass through the different stages essential for its cure. We may therefore ask ourselves whether it is that an abscess of the brain has no disposition to spontaneous cure, or whether it is that the patient does not live long enough for such a process to be accomplished? The development of a firm cyst wall would show that there is a possibility of spontaneous cure. The cyst wall exerts a protective influence, by localizing the mischief and protecting the sound from the diseased part. And experience has shown that time is only required for such protection to be very great, and for the barrier guarding the pus to become stronger and stronger.

We are next led to ask, is there anything in the condition of the pus discovered in old abscesses to show that these were in a process of cure? To our minds, there is. It is usual to find such pus in a very degenerate condition, viz. granular and fatty, which is favourable to its absorption and concretion: such changes as occur in abscesses that have undergone spontaneous cure. This is no idle question. It is simply—Is cerebral abscess necessarily a fatal and incurable disease? Practically it is, but there is nothing in its morbid anatomy to lead us to conclude that it is necessarily incurable.

DIAGNOSIS.—Cerebral abscess is inferred when there are symptoms of brain disturbance indicative of organic disease, and there are present those morbid conditions that are known to give rise to cerebral abscess, such as a discharge from the ear, nose, or chronic suppuration elsewhere, or when there is a history of a blow, or of some other acknowledged cause of the disease. No doubt that in some cases the inference proves correct, where there is evidence showing that the cerebral substance is undoubtedly diseased, and further evidence of suppuration going on in some part of the body; for here there are indications of acute brain disease, and we are led to suspect that this is due to abscess, since such causes are present as are known to produce it. With the brain, however, as with other organs, we are more often able to say that it is diseased than to say what is the precise nature of the pathological changes going on in its substance.

There may be evidence to show that a patient has chronic disease of the nose or ear, and cerebral symptoms may supervene suddenly; epileptiform seizures and other symptoms may be present, such as are seen in cases of cerebral abscess; the patient may die, and yet there may be no disease of the brain or of its membranes. In some cases, the membranes alone are diseased; in others, the brain substance is softened, without abscess. Disease of the bones of the skull—no matter whether it be fracture, syphilitic disease, or a growth—is liable to set up inflammation of the membranes of the brain, and the inflammation may spread and give rise to suppurative inflammation of the brain substance. If the patient survive six or seven weeks, an abscess may be formed; if he die in two or three weeks after acute symptoms have set in, the brain may be found softened, but without abscess. Not unfrequently death takes place before there is time for the suppurative inflammation to form an abscess.

There may be a history of injury to the head, cerebral disease may appear to have followed as a consequence, and the post-mortem examination reveal disease in the brain, but not abscess.

Injury may be followed by the formation, not of an abscess, but of a tumour, malignant disease, or by softening in the brain; or further, the disease may not be in the brain at all, but on the surface. Experience has shown that an injury to the head may produce a large cyst in the cavity of the arachnoid, and the symptoms of the case may be similar to what are seen in cases of encysted abscess.

A history of a blow on the head, followed by severe pain, loss of energy, altered manner, fits, and partial hemiplegia, occurs in abscess, but also in other cerebral diseases as well as abscess.

Cerebral symptoms associated with offensive discharge from the ear and nose, would lead one to suspect abscess in the brain, but in one of our cases there was tumour, and not abscess. The co-existence of tumour in the brain with the conditions that are known to produce abscess, makes the differential diagnosis extremely difficult. There are no pathognomonic symptoms of abscess or of tumour. It is only the different manner in which the symptoms are grouped, and the existence of those conditions that are known to produce one and not the other disease, which leads the practitioner to suspect that there may be tumour rather than abscess, or *vice versâ*.

The symptoms of abscess may differ from those of tumour in the following respects. In abscess there is often marked cachexia and great emaciation. In tumour, the patients have often no marked cachexia, even look healthy, and the body is fairly nourished, certainly not emaciated. In abscess the duration of the cerebral symptoms is generally much shorter than in tumour. The symptoms in abscess are usually either latent or acute; in tumour they are often chronic. In the latter there may be local paralysis extending over several months, which is very rare in abscess. The intracranial nerves are much more frequently affected in tumour than in abscess. Occasionally, however, a person with tumour is seen to be much emaciated. These differences may enable the practitioner, in some cases, to diagnose one condition from the other, but in neither case are these differences so constant that a certain diagnosis can be made.

An abscess may lie latent in the brain for many months, and then acute symptoms may suddenly set in, and the patient die in a few days. The same thing may take place with respect to cerebral tumour. Experience has shown that cancerous deposits also may exist in the brain without there being any decided cerebral symptoms.

Chronic encysted abscesses and tumours of the brain have many symptoms in common. An hydatid tumour; gliomatous tumour; a cyst; cancerous deposits in the brain, or any other substance acting as a foreign body, may produce pain in the head, epileptiform seizures, with or without paralysis, optic neuritis, vomiting, or gradual loss of muscular power.

We are often able to say, when there is acute persistent but variable paralysis, with pyrexia, that there is acute inflammatory softening of the brain; but whether that softening is going on around an abscess, a tumour, or a cyst, or whether excited by disease situated on the surface of the brain, we may be unable to give any exact opinion.

With respect to rigors in cases of cerebral abscess, we have already stated that they are very well marked in some instances, and may be not unlike those of ague. This symptom is not, however, peculiar to cerebral abscess. It occasionally occurs in other forms of brain disease, for instance, as gliomatous tumours or tubercle.

TREATMENT of abscess of the brain should be, by anticipation,—obviating the causes which lead to it; in chronic disease of the ear or nose, by maintaining a free exit for the discharge, no matter what the exciting cause. Rest is the most important part of the treatment, avoiding thereby both mental and mechanical excitement.

By a simple diet and quiet life, abscess may be dormant in the brain for an indefinite time.[1]

In cases where abscess follows injury to the head, surgical interference must be thought of. The principle in such cases is a mechanical one, namely, to reach the abscess and evacuate its contents, if that be thought advisable,—experience has but little to commend it.

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[1] This is, however, to be observed, that encysted abscess of the brain is fatal from changes outside the cyst of an acute kind, such as might be presumed to be preventible to a great extent. In support of this opinion we may say that, in our experience, we have known abscess lie quiet for months after a blow on the head, and the patient and the medical attendant become confident that all was well; the symptoms of lesion having slowly gone off, and yet a fatal issue be produced after a few hours' suffering by neglecting the precaution of rest and regimen. Probably such rest and care should be continued, not for months only but for years. This we say from clinical observations on the changes in the cyst of old cerebral abscess.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of Abscess.	death acute symptoms set in.	Records.
1	Male	21	Fracture of the skull from a blow of brick-bat. No depression.	Fourteen days after the accident, on admission, he was almost insensible; stertorous breathing. Right fore-arm flexed and rigid. Incontinence of urine. Skull was trephined: a tablespoonful of pus escaped: he appeared to become more sensible. Paralysed on the right side. Skin hot. Pulse from 120 to 150. He became insensible, and died nine days after the operation.	An abscess in the left hemisphere, situated immediately under the fractured portion of the skull.	Sixteen days.	Mr. Maunder kindly supplied us with the particulars of this case. Admitted Mar. 9th, 1864, into the London Hospital under the care of Mr. Maunder.
2	Male	5	A blow on the forehead. Skull fractured.	Skull was trephined, and he went out of hospital apparently well. In two months he was brought back with hemiplegia of the right side. He had had several convulsive seizures. Ten months after the injury, he was again brought to the hospital; hemiplegia persisted, and subject to fits. Optic neuritis. He died April 27th.	A large abscess occupying half the left hemisphere above the lateral ventricle. Very thick cyst.	Cannot be calculated.	This patient was under Mr. Maunder at the London Hospital.
3	Male	21	Disease of internal ear. Caries of the temporal bone.	No history.	Pus in the arachnoid. In the right hemisphere a very large abscess, size and shape of a pear, opening into the lateral ventricle. Brain around softened.	Ibid.	St. Thomas's Hospital Post-Mortem Records, 1851. Dr. Bristowe has kindly allowed us to make use of these records.
4			Necrosis of the tibia.	Fell and hurt his leg a week before admission. Abscess formed. Pyrexia set in, resembling typhoid. Stupor. Answered questions. Tongue brown and dry; no rash. Diarrhœa the last few days.	Pyæmic abscess in the brain. Three or four abscesses in the R. cerebral hemisphere. Abscesses in the lungs, spleen, and kidneys.	Ibid.	Ibid. 1858, p. 372.
5			Fractured skull.	Not given.	In the left hemisphere, a large encysted abscess. Pyæmic deposits of the lung.	Ibid.	Ibid. 1857.
6			Disease of the internal ear.		An encysted abscess in the left middle lobe.	Ibid.	Ibid.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
7	Male	10	Injury to the head. In running struck his chin, and was thrown violently on the back of his head.	Same day as the injury a convulsive fit. Left side chiefly affected. Fits repeated. In the intervals of the fits, constant burning pain over the front and right side of his head, and conscious when spoken to. The last twenty-four hours passed his urine and fæces involuntarily. Skin cool. Pulse 70. A succession of fits. Coma.	Encysted abscess in the anterior lobe of the right hemisphere, size of a walnut. Softening of the brain around. Pus in the lateral ventricles.	Had occasional convulsive seizures three months and three days.	Bartholomew Hospital Post Mortem Records, 1849. Dr. Andrew kindly allowed us to make use of these records.
8	Female	31	Abscess near the uterus.	Admitted a few hours before death, half-unconscious. Very restless, lying on her back, and often tumbling out of bed. When roused could answer rationally. Not paralysed. Died suddenly.	Two or three collections of pus in the right hemisphere; two in the right lobe of the cerebellum, pus in the ventricles. Abscesses in the liver. Ulceration of the intestine, connected with an abscess near the uterus, which had opened into the intestine.	No history except of a few hours before death.	Ibid. vol. v. 1850.
9	Male	56	Acute pneumonia, right lung. Grey hepatization and purulent infiltration.	Supposed to have caught cold whilst riding outside an omnibus. Ill three weeks before admission. On admission into hospital cough, fœtid rusty expectoration. Dulness and absence of respiratory sound over lower part of right lung. Much the same for seven days, then appeared to improve during the next eighteen days, and was able to get up. Physical signs remained.—28th day: Pain and swelling in the right leg, hardness in the course of the veins. Became worse, feverish, looked and felt ill. Complexion sallow. Delirium. Ten days before death a languid vacant expression. Slow in under-	In the brain numerous collections of greenish, rather fœtid pus—from a pin's head to a hazel-nut. One had opened into the right ventricle; one in the left lobe of the cerebellum. None in the optic thalamus, or corpus striatum, or pons.	Ill altogether about two months and ten days. Brain symptoms about ten days before he died.	Ibid. vol. v. 1850.

			pleura. A cavity at the apex of the right lung. No tubercle.	debility, with evidence of a vomica beneath the right clavicle. Strength gradually failing. Cerebral substance around abscess firm. Phlebitis in the right femoral vein. Two days before death he was found unconscious, never roused, and gradually sank.	bulged.— In the posterior part of this hemisphere several large collections of greenish yellow fœtid pus, contained in cysts of concrete pus. Size of a walnut to a pea. An abscess in the right optic thalamus. An abscess had burst into the lateral ventricles. Brain substance around congested.	toms until two days before death.	1850.
11	Female	20	Old empyema in the left pleura. No tubercle.	Admitted two months before death with a copious, offensive, greenish, purulent expectoration. Physical signs: complete dulness all down left side behind; amphoric respiration and pectoriloquy. She improved, and able to walk about. Physical signs remained the same. Three weeks before her death she began to complain of headache. Next day an epileptic seizure. Three days afterwards another. Was pretty well for a week; then another fit, followed by as many as eighteen in twenty-four hours, and was almost unconscious in the intervals. Following day became more conscious; fits returned, and recurred during the next six days; then she died.	Right hemisphere flattened. An encysted abscess in the middle of this hemisphere, and it had burst. Cyst walls firm, dense. A wineglassful of offensive pus of a dirty, greenish colour, streaked with brown. Brain substance soft and pulpy. A large suppurating cavity in the left pleura. No tubercle. Rest of the body not permitted to be examined.	Three weeks before death.	Ibid. vol. v. 1851.
12	Male	—	Unknown.	Admitted for dyspepsia, nausea, and sense of oppression. This continued for fourteen days, then disappeared. About three months afterwards he was taciturn, heavy, and silent; answered questions rationally. Urine passed involuntarily. Ten days afterwards pain in the crown of the head. Fifteen days after this he became comatose, and died. No paralysis. Sickness was the early and constant symptom.	Encysted abscess in both hemispheres, size of an orange; pus inodorous; brain substance around, soft. Other organs healthy.	Acute symptoms began about fifteen days before death.	*The Lancet*, July 12th, 1862 by Dr. Leith Adams.
13	Male	47	A fall on his head a few months before death.	Seized with convulsions, followed by hemiplegia. Delirious. Sank into coma and died.	An abscess in the right hemisphere, opening into the right lateral ventricle.	Duration of acute symptoms, a week.	*Med. Times and Gaz.* 1861, p. 196. Under the care of Dr. Tuke, York.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
14	Male	66	Discharge from the ear for several years.	Deafness on one side. Went to bed as well as usual; next morning paralysis on one side of the face, also ptosis. Paralysis persisted for some days; became giddy; had severe rigors; drowsy; continually dozing unless spoken to; delirious at times; face flushed; head hot; tongue brown. He had an attack of convulsions. Gradually sank and died.	In the centre of the right cerebral hemisphere, a large abscess.	Duration of acute symptoms, twenty-three days after admission into the hospital.	*Med. Times and Gazette*, 1863. Under the care of Dr. Baly.
15	Male	16	Injury to the head. No fracture. Bone laid bare.	Fell into a ship's hold. Had been subject to fits. Had one about half an hour after admission. For nine days doing well, then drowsiness and stupor set in. Urine passed involuntarily. Became greatly emaciated. Skull trephined. No relief. He died forty-seven days after the injury.	An abscess in the left anterior lobe contained an ounce of pus. Rest of the brain healthy. No fracture. No other organ examined.	Forty-seven days.	Ibid. 1862, p. 267. Under the care of Dr. Lunn, Hull Infirmary.
16		18	Disease of the temporal bone.	None, except convulsions immediately before death.	A large cerebral abscess.	No acute symptoms until just before death.	Ibid. Dec. 1861. Under the care of Mons. Richel.
17			Scalp wound. Bone dead.	Rapid emaciation. Heat of skin. Appetite was good. On the 28th day of admission allowed to get up. Rigors in the night, unconscious next morning, but could be roused. Trephined. A little pus escaped. Died the 30th day.	Abscess in the left hemisphere, size of a hen's egg. No abscess elsewhere.	The rigors set in two days before death.	Ibid. Dec. 8th, 1860. Under the care of Mr. Paget.
18	Male	31	A nasal polypus by pressure obliterated the trunk of the internal carotid artery. Absorp-	Admitted for profuse epistaxis. A few days afterwards an epileptic seizure which left him hemiplegic on the left side. Ten days afterwards another fit. Thirteen hours afterwards coma and death.	Three abscesses in the right cerebral hemisphere. Brain substance on that side much softer than on the opposite.	Some days; the number not given.	Ibid. June 19th, 1858. Under the care of Mr. Simon.

			sphenoid bone.				
19	Male	Adult	No history of injury to the skull. No other internal abscess mentioned. No mention of the ear or nose.	Complained of headache. Generally unwell. Had had pains in his head for four or five days. Tongue brown and dry. Thirsty. Eyes suffused. Became semi-comatose. Pulse intermittent. Died five days afterwards. Body not emaciated.	A large abscess in the anterior part of the right hemisphere. Body not emaciated.	According to the Record, pain in the head about nineteen days before death.	*Medical Times and Gazette*, Feb. 21st. Under Mr. Grant, 54th Regiment.
20	Male	34	Supposed cause injury to the head.	Pain in the head and in his teeth and left side. Tooth extracted. Fifteen days afterwards said to have had an attack of general convulsions. Convulsions repeated. Speech was slow. Complexion became yellow. Vomiting. Facial paralysis on the right side. The patient died with signs of compression five months after the first appearance of symptoms.	Four encysted abscesses, two the size of a walnut, and two the size of a hazel-nut, in the left middle lobe. Brain around the abscesses, soft.	Doubtful.	*Archives générales de Médecine*, 1860, p. 672. Professor Griesinger, of Tübingen.
21	Male	20	Disease of the ear.	Had discharge from his ear nearly four years. On the 22nd December, head and neck rigidly bent back, and spine curved. Some rotatory movements of the head. On attempting to draw his head forward it elicited an expression of great pain. Was unable to swallow. Next day he suddenly became asphyxiated, and died.	An abscess the size of a walnut in the pons Varolii, which had burst.		*Pathological Transactions*, vol. xi. Recorded by Dr. Down.
22	Male	22	Disease of the tympanum. No caries of the bone.	Sore throat for one week, and became generally ill. A discharge from the ear. Redness and swelling in the throat. Great depression. April 25th admitted, and May 2nd, rigors; great prostration. Two days afterwards pain in the right side. Respiration quick. He became heavy and stupid, passed into a semi-comatose condition, and died.	An abscess the size of a hen's egg, in the middle right lobe of the brain. Abscess as large as a walnut in the right lobe of the cerebellum.		Ibid. vol. xv. Recorded by Dr. Dickinson. Patient under Dr. Barclay, St. George's Hospital.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
23	Female	41	Suppuration of the right internal ear.	Discharge from the ear for several years. Admitted into Bartholomew's Hospital one month before death. Loss of power of right half of face. Some spasmodic pain. Constant pain right side of head. Hyperæsthesia on the right. Became drowsy, semi-comatose, conscious when spoken to, and conversed with her friends the day before she died. No paralysis or irregular movements of the limbs. Could feed herself and stand up.	Abscess in the middle of the right lobe of the cerebellum; it communicated directly with the diseased portion of the temporal bone. The abscess encysted.	About forty-two days.	St. Bartholomew Hospital Post-Mortem Records, vol. v.
24	Male	23	Caries of the temporal bone.	Admitted the day before his death, complaining of great pain in his head, especially in the back of his head. It felt like a coal of fire. Deaf on the right side and troubled with ear-ache since he had scarlet fever some years before. Occasionally vomited. Head hot. His illness commenced eleven days before admission, with rigor, followed by constant pain in the head. The night of admission, pain agonizing. He screamed with pain. Quite conscious. Pain continued up to the time of his death. An hour before he died he became quiet. No paralysis. No irregular movements. He walked to the Hospital the day before he died.	In the right lobe of the cerebellum one abscess the size of a walnut. The abscess had opened on the surface of the brain. Caries of the right temporal bone. Chronic ulcer of the stomach.	About fifteen days.	Ibid. vol. viii.
25	Male	25	Chronic disease of tympanum.	Two or three restless nights. Severe frontal headache. On the fourth day vertigo and delirium, with slow pulse. Efforts to vomit. Fifth day, cerebral oppression. 6th, paralytic weakness of the left side. 7th, coma; death.	Acute abscess in middle lobe of cerebrum on right side. Dura mater sloughing. Petrous bone carious.	About seven days.	Recorded by Dr. Gull, Guy's Hospital Reports, vol. viii. 3rd Series.
26	Female	23	Chronic disease of tympanum.	Severe headache, principally over the right side of the head. Pain in the right ear,	Diffused suppuration, and acute sloughing of the	About seventeen days.	Ibid.

				Paroxysms of extreme restlessness. Cerebral oppression. Death on the 17th day. No convulsion nor paralysis throughout.	sphere. Dura mater over roof of tympanum sloughing. Bone carious.		
27	Female	20	Disease of the tympanum following a blow on the ear.	Paralysis of right seventh nerve, and discharge from the ear, and headache after a blow. After three weeks headache increased, referred to forehead and occiput. Pain on moving the neck. Rigors, nausea, vomiting, sweating. No delirium. Death from syncope on the 4th day after the increase of the headache.	Abscess in cerebellum. Inflammation of the vein of the aquæductus vestibuli.	About twelve days.	Ibid.
28	Male	43	Chronic disease of the mucous membrane of nose.	Sudden lightness in the head, followed, after a short interval, by convulsion and insensibility, recovery, and a second convulsion the same day. On the 3rd day headache, increasing to great intensity on the 5th; referred to the right side of the forehead, right temple, and occiput. No delirium. Death on the 8th day, in coma.	Acute abscess in middle lobe of cerebrum on the right side.	Eight days.	Ibid.
29	Male	13	Chronic disease of the ear.	Languor for some days. Syncopal seizure. Convulsions with insensibility, relieved by a discharge of pus from the right ear. Following day severe headache from the forehead to the vertex. Nausea. Delirium at night. 6th day, return of convulsions, with insensibility. Intense pain and cramp in left leg. Death in rather sudden coma, 15th day.	Abscess, containing about an ounce and a half of pus under the posterior lobe of right hemisphere of the cerebrum, between it and the tentorium. Caries of petrous bone.	Fifteen days.	Ibid.
30	Male	25	Dysentery. Chronic abscesses in liver. Recent abscess in lung.	Epileptiform convulsion, followed by apoplectic symptoms lasting several hours. Only partial recovery of memory after ten days. On the 14th, vomiting; increased cerebral oppression. At beginning of 4th week sank into sudden coma, with paralysis of right side. Death on 26th day.	Undefined abscess in posterior lobe of cerebrum, on left side.	Twenty-six days.	Ibid.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
31	Male	43	Cause not found.	Symptoms of hepatic derangement. During *apparent convalescence*, sudden severe neuralgic pain over left eye. Restlessness. On 3rd day defective articulation. Delirium. Gradually increasing coma. Death on 10th day.	Abscess, without definite walls, in anterior lobe of left hemisphere, near the surface. A second abscess, encysted, in the middle lobe on the right side. This had burst through the optic thalamus into the lateral ventricle.	Ten days.	Recorded by Dr. Gull.
32	Male	46	Chronic abscess in sheath of left rectus abdominis muscle.	Under care for chronic abscess in abdominal walls. Rigor; drowsiness Great muscular debility, especially marked on the left side. Partial paralysis of right side of face. Difficult deglutition; semi-coma. Death on 6th day.	Five abscesses, in different parts of the medullary substance of the right cerebral hemisphere. Abscess in left hemisphere of cerebellum. Abscesses not encysted.	Six days.	Ibid.
33	Male	45	Suppuration of mesenteric glands after ileitis. Abscess in spleen and kidney; not encysted.	Febrile symptoms; frequent rigors; pain, supposed to be neuralgic, in left side of head; sudden hemiplegia, without loss of consciousness; drowsiness. No delirium. Death by general exhaustion at end of three weeks.	Numerous abscesses, not encysted, scattered through the medullary substance of hemispheres; one in the right corpus striatum.	Three weeks.	Ibid.
34	Male	16	Disease in the orbit.	Phlegmonous inflammation of upper lip, extending to the right orbit. Abscess behind the globe relieved by puncture. Pain of an intermittent character over *left* side of the head, extending along the lower jaw and behind the ear. *Apparent convalescence.* Sudden dull heavy pain in head, with vertigo. General convulsions, coma, and death, in about five hours.	Abscess, not encysted, occupying two-thirds of the middle lobe of the brain on the right side. Suppuration in orbit of the same side. Sloughing dura mater.	Cannot be calculated.	Ibid.

35	Male	16	Fall on the back of the head.	Symptoms of typhus in a mild form. Headache, relieved by leeches. *Apparent convalescence.* Sudden insensibility, followed by a succession of epileptiform convulsions. Partial recovery of consciousness. Return of convulsions, followed by hemiplegia of right side. Death in coma, after ten weeks.	Large encysted abscess in middle lobe of cerebrum on the left side.	Ten weeks.	Recorded by Dr. Gull.
36	Female	17	Cirrhosis of left lung, with large suppurating cavities (dilated bronchial tubes?).	During convalescence from variola, sudden maniacal delirium. Rigor; headache; drowsiness; paralysis of left arm. On 4th and 5th day severe pain in left leg. On 6th, the leg was incessantly moved up and down in bed; sudden coma, and death about noon the same day.	An encysted abscess, which had burst through the right optic thalamus into the lateral ventricle. Two smaller encysted abscesses in posterior third of left hemisphere.	Eight days.	Ibid.
37	Male	34	Suppurating cavity in right lung during three years, after pleuropneumonia.	Sudden seizure with vertigo, faintness, and loss of power on right side. No unconsciousness. Complete recovery, and good health for eight months. Sudden clonic spasm of right arm, lasting a few minutes, and returning several times within a few hours. No loss of consciousness. Following day, spasm of arm, beginning as before, but the attack soon became epileptiform. 4th day, return of epileptiform convulsions, followed by partial hemiplegia of right side, gradually becoming complete. Death after three weeks from second seizure.	Large encysted abscess in posterior lobe of left cerebral hemisphere.	Three weeks from second seizure.	Ibid.
38	Male	23	Chronic disease of right pleura, with external fistulous opening. Dilated bronchial tubes.	Sudden seizure, with only partial loss of consciousness. A second seizure the same day. After a month, a third seizure, followed by impaired memory and general cerebral oppression. In the interval of the seizures, headache and occasional vomiting. Death at the end of three months.	Encysted abscess in posterior lobe of left cerebral hemisphere.	Three months.	Ibid.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
39	Male	40	Fall on pavement; contrecoup, a year before distinct symptoms of cerebral disease.	Severe headache, principally frontal; gradual impairment of memory; transient attacks of loss of sight, sometimes with unconsciousness. These symptoms for a year. Subsequently, total blindness; continued headache; partial hemiplegia of left side. Frequent seizures, with general loss of muscular power. Drowsiness; semi-coma. Death 14 months after the beginning of the symptoms.	Two large inflammatory cysts, with surrounding solid exudation in the anterior lobe of right cerebral hemisphere.	Fourteen months from the commencement of chronic cerebral symptoms.	Recorded by Dr. Gull.
40	Male	42	Chronic disease of mucous membrane of nose.	Mucous discharges from nose for an uncertain period. Vertigo and headache five weeks. Seizure in bed at night; insensibility; paralysis of left arm and leg. Death after twelve days.	Softening and ulceration of the convolutions of the under surface of anterior lobe of right hemisphere, extending backwards to the fissure of Sylvius and inferior surface of corpus striatum.	Twelve days.	Ibid.
41	Male	35	Syphilitic disease of the bones of the head. Caries of the cranium and perforation of the dura mater.	Had syphilis five years before. Was in the hospital three years before for diseased bones of the head. Pieces of dead bone removed. Three weeks before admission was delirious. On admission very weak; drowsy; vacant expression; fits of forgetfulness. Became more drowsy, and died the day after admission.	Anterior two-thirds of the right cerebral hemisphere converted into a collection of fœtid pus. Entire brain somewhat softened.	About three weeks and two days.	Recorded by Dr. Ogle, *British and Foreign Medico-Chirurgical Review*, No. lxx. p. 464.
42	Male	26	Pneumonia. Purulent infiltration.	Admitted with pneumonia in the left side. Had headache for months. Subject to involuntary spasm of the right arm. Headache continued. Subsequently had a fit. The right side chiefly affected. He remained hemiplegic on the left side. Vomited. Strabismus. Could not swallow. Skin became hot and dry. Twitching of the right arm and leg, and loss of sensibility. Occasional to-and-fro movement in the left arm. Partial paralysis right side of face. Right con-	Inferior and posterior part of middle lobe of the left hemisphere softened, and near the surface a cavity the size of a hazel-nut, lined by soft fibrinous material, and containing pus. The sinuses lateral, superior, longitudinal, and petrosal, as far as the internal jugular vein, were filled with firm blood co-	Cannot be calculated.	Ibid. Case 85.

				became opaque. Evacuations passed involuntarily. Several general convulsive attacks. Sank and died.	broken down into grumous fluid. Many similar clots in the superficial vessels between the convolutions.		
43	Female	46	"A fall."	Thirteen weeks before admission had a fall; ill ever since. Headache and vomiting and bleeding at the nose after the accident. Three weeks before admission headache and sickness constant and severe, and gradual decline of mental vigour. On admission partly delirious; tongue dirty; pulse very weak. Evacuations passed involuntarily. Died of exhaustion on the 9th day of admission.	Body much emaciated. Skull thick. Membranes healthy. Convolutions much flattened. Lower part of the posterior lobe on the right side, a large loose clot of apparently fresh blood; brain around broken down. Left side of the cerebellum a cavity equal to a hazel-nut in size, containing a quantity of healthy-looking pus. Walls of the cavity easily dislodged from the substance of the cerebellum, which was softened around. Arteries at the base atheromatous.	Died fourteen weeks two days after the injury. Headache and vomiting followed the fall. Does not say the precise time the headache set in.	Dr. Ogle. Case 86.
44	Male	Middle age	No history could be obtained.	On admission, great pain in the back of his neck, and in the head. Died in great suffering in the course of a few hours.	In the anterior part of the right corpus striatum an encysted abscess; burst into ventricle. Other organs of the body healthy.	Cannot be calculated.	Dr. Ogle. Case 87.
45	Female	30	No mention of abscess on any other part of the body. The ear not examined.	Intemperate prostitute. Not known to have had any cerebral symptoms. Comatose twelve hours before admission. Motions passed involuntarily. Left arm paralysed. Sensibility remained. Biceps of the left arm strongly contracted. Patient thus a week. Breathing became impaired, and died 7th day afterwards.	Abscess in the right cerebral hemisphere. Large and containing greenish-yellow offensive pus, extending from the anterior border of the corpus striatum almost to the posterior part of the hemisphere. Separated from the brain's surface by a thin layer of condensed cerebral matter. Arachnoid healthy; sinuses and dura mater, the same. Temporal bones not examined. Other organs healthy.	About seven days.	Case 88.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
46	Male	16	Compound fracture of right parietal bone of the skull.	Fell eleven feet. Admitted half stupid. Capable of answering questions. Scalp wound, and depressed fracture. Hernia cerebri. No pain on admission. Slight pain came on later. Tongue became white. Incision was made into the wound. Much discharge, mixed with brain substance. Went on well until 14th day, when suddenly became unconscious. Stertor. Dilatation of the pupil. Pulse fell fifty-two, and he died.	Fracture of the right parietal bone. Between dura mater and bone, flakes of coagulated blood. Corresponding to this, recent fibrin; on breaking through it a large quantity of creamy pus, escaped from an abscess so large that it passed into the anterior and posterior lobe of the hemisphere. All of the outer part of the middle lobe destroyed. Separated from the lateral ventricle only by the lining membrane. Brain elsewhere natural.	Died fourteen days after admission.	Dr. Ogle. Case 91.
47	Female	44		Two weeks before admission, much exposed to the sun's rays. Felt giddy. Soon after fell into some water. Much frightened. Manner became changed. Miscalled objects. Could not be understood. Difficulty in swallowing. No febrile symptoms. Often raised her hand to her head. Complained of feeling ill. Tongue furred. Apathetic. Right arm spasmodically flexed, and could not be straightened. Right pupil dilated.	Membrane congested. Anterior two-thirds of the left cerebral hemisphere broken down into a mass of greenish purulent matter. In the middle lobe of the same a collection of yellow pus. Other organs presented nothing unusual. Pus in left ventricle.	Cannot be calculated.	Dr. Ogle. Case 92.
48	Male	44	Scalp wounds. Arachnitis.	Pulled to the ground, and three or four scalp wounds produced over the right part of the forehead, penetrating to the bone. Went on well. In a day or two	Outer surface of cranium roughened. Pus between dura mater and bone, opposite one of the scalp	Died on the 18th day of admission.	Dr. Ogle.

				rigors came on. Pulse 108. Skin dry. Tongue creamy. Rigors returned. Pain felt in the ankles and shoulders. He sank and died May 25.	wounds. Pus in arachnoid. At one part dura mater ulcerated, and in the brain substance beneath was an abscess. The entire anterior lobe on the right side softened. Superior longitudinal sinuses contained purulent fluid. Lungs, scrofulous deposits.		Case 89.
49	Male	25	Fracture of the occipital bone.	While drunk sustained a compound fracture of the occipital bone. Headache; restlessness; slight want of power in the muscles of the right side of the face. Pupils dilated. Went on the same until 16th day, when seized with twitchings of the muscles of the face. Coma set in, and he died on the 16th day of admission.	Upper surface of both hemispheres smeared over with blood; white portion of the right anterior lobe bruised, and between it and the surface of the brain, an abscess near the orbital plate of the ethmoid bone. Pia mater thickened and congested. Lateral ventricles distended with purulent fluid, and lining opaque. Recent lymph in sub-arachnoid spaces.	Fifteen days after admission. Headache came on two days after admission, and died thirteen days afterwards.	Dr. Ogle. Case 90.
50	Male	79	Carcinomatous ulceration of the integuments and bones of the face and walls of the right orbit.	During the whole of his disease there were no symptoms to indicate brain disease.	Ulceration and perforation of the sphenoid bone and dura mater. Adjoining this near the convolutions an abscess in the substance of the right cerebral hemisphere, and it had contained a large quantity of pus which had escaped through the apertures in the dura mater and bone.	No symptoms.	Recorded by Dr. Ogle. Case 76.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of Abscess.	How long before death acute symptoms set in.	Records.
51	Male	25	Abscess in the liver, communicating with the surface of the body.	Five weeks before admission had pain in the right side of abdomen. It abated and then recurred. Was obliged to go to bed. Hot skin. Pain in the right side of belly, and some vomiting. Abdomen distended; walls hard and contracted. He much improved. Suddenly attacked with chills and heats. Got thinner and weaker. An abscess pointed and discharged at the umbilicus. Pus became tinged with bile. Pus in the stools. Rigors and diarrhœa came on. Delirium. Coma. Died in a typhoid state. No paralytic symptoms.	Numbers of minute abscesses in every part of the brain, in the white as well as in the grey matter, in the corpus striatum, optic thalamus, and pons Varolii, all smaller than a pea; surrounding brain softened. Secondary abscesses in the lungs. Several abscesses in the liver. Ulcers in the large intestine.	Cannot be estimated. No characteristic cerebral symptoms.	Recorded by Dr. Ogle. Case 77.
52	Male	15	Superficial scalp wound of the forehead, caused by fall.	Drowsiness supervened, but he improved. Eleven days after admission, great headache. Pulse quick. 14th day, two attacks of convulsions. Delirium and pain came on. Right pupil insensible to light; dilated. Later, passed evacuations involuntarily. Spectral illusions. Answered questions sensibly. Died on the 26th day after admission.	An abscess in the left cerebral hemisphere, anterior part. Lateral ventricles full of pus. Fracture of the left frontal bone. Scalp was quite healed.	Fifteen days before death.	Recorded by Dr. Ogle. Case 78.
53	Male	25	A blow on the head. A scalp wound without fracture.	Blow, left side of head. Severe headache three weeks afterwards, followed by rigors; sweating; articulation became affected, and slight hemiplegia on the right side supervened. Admitted into St. George's Hospital, Nov. 24th, 1847. Bone of the scalp exposed. Symptoms relieved. Complete hemiplegia came on. Skull trephined. Foul pus escaped through a sloughy hole in the dura mater. Died 34 days after first symptoms set in.	No fracture of the skull. Sloughy opening in the dura mater, leading to an abscess in the posterior part of the left cerebral hemisphere. Walls firm and distinct. Surrounding brain substance pulpy and of a yellow colour. Other organs healthy. Lungs congested.	Thirty-four days	Recorded by Dr. Ogle. Case 79.
54	Male	35	Had a cough	February 20th, numbness of the right arm	A circumscribed abscess, the	Eighteen days.	Recorded by

			years, and un-able to work; but no proof of old - standing lung disease.	that side. 24th, a fit. Partially un-conscious. Hemiplegia became complete. Much headache; mind unaffected. Vomiting came on. Great prostration. Gradually sank, and died 18 days after the numbness in the arm.	left cerebral hemisphere, middle lobe, lined by a false membrane: contained dirty green pus. Surrounding brain softened.		Case 80.
55	Male	26	No mention of abscess elsewhere. Idiopathic?	On February 23rd, previously in good health, he was seized with hemiplegia of the left side. No loss of consciousness. Sensibility of skin not much affected. On admission, left arm most paralysed. Paralysis left side of the face. Pain in the head. Under treatment he improved. Recovered somewhat the use of his arm. 25 days after admission, rigors set in—shook like a person in ague—followed by a fit and coma. Died next day.	Sub-arachnoid tissue infiltrated with pus. A circumscribed, encysted abscess above the roof of the right lateral ventricle. Cyst wall was one-twelfth of an inch thick. Contained greenish fetid pus. Surrounding brain softened. Lateral ventricles filled with pus.	Thirty days.	Recorded by Dr. Ogle. Case 81.
56	Female	53	Phagedænic wound following the removal of scirrhus of breast.	Admitted August 31st. September 8th she had an apoplectic attack, followed by coma, loss of power in the right arm and leg, and left side of face. Difficult articulation. The arm partially recovered. Bed-sores came on, and she died December 1st; that is, 53 days after the cerebral symptoms set in.	The left optic thalamus contained a quantity of purulent deposit, very much softened, to the extent of a threepenny piece, of an ochry colour. Left corpus striatum and neighbouring parts of the brain extensively softened. An abscess behind the cæcum; the latter ulcerated.	Fifty-three days.	Recorded by Dr. Ogle. Case 82.
57	Male	23	Abscess of the lung? Pyæmic abscess in the brain.	Admitted March 13th, 1856. Been ill three weeks with feverishness, shivering, and languor. On admission, rapidly passed into a typhoid state. No rose-spots. Had also pain in the head, and lost the use of his right arm. Sensibility partially destroyed. Pulse 88. Sickness came on. Unable to answer rationally. Tongue dry and raw. Right arm became less paralysed. A succession of fits, and died March 22nd.	Several abscesses in the substance of the brain. Two in the right hemisphere, about an inch from the surface. A largish one in the centre of the left hemisphere; also two smaller ones. Abscesses in the left hippocampus major, left optic thalamus, and cerebellum. An abscess in the right lung.	Thirty days.	Recorded by Dr. Ogle. Case 83.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
58	Male	35	Abscess following amputation of the arm. Necrosis of the ulna. An abscess in the deltoid muscle.	After amputation of the fore-arm, he left the hospital; got frequently drunk. He was re-admitted October 31st, 1860, was shivering, sweating, and vomiting. The rigors recurred daily, almost at the same hour, reminding one of ague. An abscess formed in one of the buttocks. Cough, pain in the chest, bloody expectoration came on. Surface became yellow. He died 27 days after admission.	A collection of purulent matter, the size of a filbert, was found in the lower part of the left middle lobe, not encysted. Secondary abscess in the lung. Other organs healthy.	Had symptoms twenty-seven days before he died.	Recorded by Dr. Ogle. Case 93.
59	Male	28	Disease of the ear.	Some years subject to occasional discharge from the left ear. Three weeks before admission had a blow on the head from the edge of a door. Followed by intense pain at the seat of the blow, and subsequently over the whole head. Week after, delirious, and frequent attempts at self-destruction, and 25 days after became comatose and died.	A large abscess in the anterior and middle lobes of the left hemisphere, the walls of which were in a state of gangrene. Abscess communicated with the tympanum by an ulcerated opening in the petrous portion of the temporal bone.	Symptoms set in twenty-five days before death.	Recorded by Dr. Ogle. Case 97.
60	Male	27	Caries of the internal ear, and diseased lateral sinus.	Six months had purulent discharge from the right ear, deafness, and pain in the head. It became fetid and copious. A month afterwards, paralysis of the right side of the face, and tendency to stupor. Phthisical symptoms. Extensive hæmorrhage from the ear caused death.	Temporal bone carious. Dura-mater sloughing. Under surface of the right middle cerebral lobe, sloughy. Medullary matter softened and contained foul purulent fluid. Lateral sinus connected with the carious temporal bone much inflamed, almost sloughy. Foul pus in the cavity of tympanum. Scrofulous tubercles in the lungs and peritoneum. Bowels adherent.	Duration of symptoms, three months and nine days.	Recorded by Dr. Ogle. Case 98.
61	Male	8	Caries of the temporal bone.	Except slight discharge from the left ear since quite young, has been in good health up to six weeks ago, when he had	Cerebral convolutions flattened. Brain generally, very vascular. In outer part	Twenty-seven days before death.	Recorded by Dr. Ogle.

				vomiting. A second fit seven days afterwards. Left him with pain and discharge from his ear, and passing his motions involuntarily. Been "silly" since the fits. On admission, paralysis of the left upper eyelid. Mouth drawn to the left, and tongue turned to the right. Limbs all weak, but perfect power over them. Complained of nothing but of twinges of pain in his left ear, and deafness. Articulation imperfect. Became dull and drowsy. Semi-comatose. Then a convulsive attack. Coma. Several fits followed. Coma, involuntary evacuations, and death.	sphere, including the greater part of the middle lobe, a large abscess. Walls firm, tough, and a quarter of an inch thick. Lined with a blackish sloughy membrane, containing 6 ounces of fetid pus. Surrounding brain soft and pulpy. It approached quite to the surface of the brain, and was adherent to the dura mater, covering the petrous portion of the temporal bone. At this spot a communication existed between the abscess and internal ear. Ulcerated opening in the dura mater. Cavity of the tympanum full of pus.		Case 99.
62	Female	26	Right temporal bone.	Six weeks before admission, subject to boils. Two weeks before admission, pain in and purulent discharge from the right ear. Three days before admission she became delirious. When admitted, great maniacal excitement; obliged to be confined in a strait jacket. Three days afterwards, slight attack of opisthotonos. Retention of urine. Incontinence of fæces. Sank into coma, and died.	Purulent fluid in the right arachnoid. Convolutions generally flattened. Brain throughout softened. In ventricles six ounces of semi-purulent fluid. Lining membrane vascular. Septum lucidum quite destroyed. Fornix diffluent. Under surface of the middle cerebral lobe ash-grey colour. In it an abscess size of walnut, lined by a thick cyst of organized lymph, and containing 1oz. of milk-white pus. Fibrinous exudation between the surface and the bone; the latter slightly diseased and having an aperture leading to the tympanum, the latter ulcerated. Full of pus.	Fifteen days before death.	Recorded by Dr. Ogle. Case 100.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
63	Female	51	Caries of temporal bone. Coagulum in sinuses.	Admitted with cough, pain in limbs, neck and throat, as if "a cold." Pulse quick, and a cachectic look. Purulent discharge from the left ear. Recovered sufficiently to leave the hospital, but had slight headache and discharge from the ear. Next day had a fit, and day following insensible. Again admitted, incoherent, partially conscious. Pulse full; tongue furred. Very drowsy. Urine passed involuntarily. Gradually sank, and died.	Dura mater ulcerated over a spot corresponding to a carious opening in the temporal bone. Pia mater highly congested and ecchymosed. Whole brain much softened, especially the left cerebral hemisphere, and in this a collection of pus, in a firm, distinct cyst. Purulent fluid in the ventricle. Septum broken down.	Fifty-three days before death.	Recorded by Dr. Ogle. Case 101.
64	Female	23	Disease of the ear, following scarlet fever.	Subject to leucorrhœa; much headache for two weeks before admission. Had scarlet fever when a child, *seen double* ever since at times, and had discharge from the ear. Headache ever since a child. On admission, rambling. Pulse quick and soft. Never any rigors. Thirst. A series of epileptic attacks. Slight convulsion; strabismus. Complained of great pain in the head, feeling "as if her eyes were shooting out." Much pain down the back and cardiac region. Pyrexia. Intense agony and screaming. Remained sensible until her death, which occurred without further supervention of convulsions.	Pus in the scalp over right temporal bone, with diplöe full of pus. Sloughy dura mater. Pus between the latter and bone in the right, middle, and posterior fossæ. At this part, in the substance of the brain, an abscess, the size of a walnut. Surrounding brain soft and vascular. Left lateral sinus, coagulum of fibrin and blood.	Twenty-nine days before death.	Recorded by Dr. Ogle. Case 102.
65	Female	54	Necrosis of the right temporal bone.	Six months before admission had a fit after a hearty meal, but no convulsive movement. Soon recovered to some extent, but his mind remained affected, and he had attempted suicide. Frequent pains in the forehead, but no loss of muscular power. On admission, stupified. Soon became conscious. Much pain in	A very large abscess occupied the whole of the middle lobe of the right hemisphere down to the base, where the membranes were united together, and adherent to the petrous portion of the right	Cannot be calculated.	Recorded by Dr. Ogle. Case 104.

				Loss of consciousness, but not convulsed. Loss of sensibility. Remained comatose. Stertor, and died in a convulsive attack.	serum.		
66	Female	7	Disease of the left ear.	On admission, discharge from the left ear. Enlarged cervical glands, and great debility. Scarlet fever two years ago. Four months a fetid discharge from the left ear. On admission complete absence of fever. She much improved under treatment. One day, after syringing, she had a fit. She complained of pain in her head. She recognised people although the convulsive attacks persisted. Never wandered in her mind. Fits continued, and she died.	Surface of the brain much flattened. Much clear fluid in the lateral ventricles. Septum lucidum softened. Upper part of the left lobe of the cerebellum contained half an ounce of greenish pus in an irregular cavity. It communicated with the surface of the brain by an orifice corresponding to the internal auditory foramen of the left temporal bone, which was found to contain a quantity of pus. Surface of the temporal bone natural. Membrana tympani absent and bone of external auditory foramen exposed.	Undoubted cerebral symptoms four days before death.	Recorded by Dr. Ogle. Case 105.
67	Male		Disease of the internal ear.	Admitted with sore throat, of a week's standing, and extreme difficulty in swallowing. Discharge from the right ear. *No ulceration* at the back of the throat. Offensive breath and foul tongue. Improved. Discharge from the ear ceased rather suddenly. Shortly after severe rigors and collapse supervened. Two days later, severe pain at the right side and hurried breathing came on. Became stupid, heavy, and comatose, and died.	Ulceration of the dura mater over the anterior surface of right temporal bone. Pus between the membrane and the bone, also in the lateral sinus and internal jugular vein. Brain adherent. An ulcerated opening led into an abscess in the right middle lobe, the size of a hen's egg, full of pus. In the right lobe of the cerebellum was an abscess the size of a walnut, with an orifice, and attached to the dura mater. Pyæmic abscess in the lung. The temporal bone exposed on passing a probe into the external meatus.	Cannot be calculated.	Recorded by Dr. Ogle. Case 106.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*	*How long before death acute symptoms set in.*	*Records.*
68	Female	26	Disease of the mucous membrane of the tympanum.	January 26th, 1846, complained of earache on the right side. Had a discharge from the right ear since an attack of measles when a child: discharge very offensive at times. The last twelve or sixteen months had suffered from headaches, occasional forgetfulness, and giddiness. Pain increased; came on in paroxysms. February 17th, she was partially insensible, became comatose, and died twenty three days after the pain commenced.	An abscess occupied the whole of the upper part of the right cerebral hemisphere. Surrounding brain healthy. Lymph on the dura mater, covering petrous bone; membrane covering squamous bone was thick and detached. Tympanic, mucous membrane, and that of the mastoid cells, was thick and soft, and covered with cheesy matter.	Twenty-three days.	Recorded by Mr. Toynbee, "Diseases of the Ear," p. 257. 1st Edition.
69	Female	9½	Caries of the upper wall of tympanum. Arachnitis.	Had measles when a child; offensive discharge from the ear, and occasional pain ever since. May 5th, seized with symptoms of fever. Constant vomiting. On the 7th seemed well. 8th, the bad symptoms reappeared. 10th, excruciating pain in the ear. Slight paralysis in the left side of face. Became comatose, and died twelve days after the appearance of first symptoms.	Pus in the left arachnoid. In the interior of the left middle cerebral lobe was an abscess the size of a hen's egg. Contained fetid pus. Caries in upper wall of tympanum. Dura mater ulcerated.	Twelve days.	Recorded by Mr. Toynbee, p. 259.
70	Female	12	Caries of the temporal bone: death following a blow on the head.	July 2nd, 1850, received a violent blow on the head. 3rd, had violent pain in the head, chiefly in the temporal region. Severe febrile symptoms. Symptoms increased. An abscess formed beneath the temporal muscle. Coma ensued. Death twenty-two days after the injury.	Dura mater lining the squamous bone thickened and adherent to the bone and arachnoid, and a portion of the brain. An abscess in the middle lobe contained four ounces of pus. Petrous bone was diseased.	Twenty-two days.	Recorded by Mr. Toynbee, p. 308.
71	Male	12	Caries of the right lateral sulcus. Catarrhal inflam-	Had scarlet fever two years ago. Discharge from the ear since. February 13th, 1854, rigors and general malaise Pyrexia. 15th, abscess behind the ear. Slight stupor.	An abscess was found in the middle lobe of cerebrum. Pus between the diseased mastoid process and dura	Thirty-one days.	Recorded by Mr. Toynbee, p. 322.

			membrane lining the mastoid cells. Retention of the discharge from the right ear.	23rd, the pain and feverishness returned. Drowsiness increased. Became weaker. Urine and fæces passed involuntarily. March 4th, two severe rigors. Severe pain in the head. 6th, pain increased. The following seven days suffered much less pain. 15th, vomited much. 16th, suddenly became convulsed and died.	behind the ear.		
72	Male	35	Caries of the mastoid cells. Polypi in the external meatus.	Suffered from frequent severe ear-ache for some years, with discharge. Five weeks before death a polypus removed from the external meatus, soon followed by great pain at the back of the head, neck, and shoulder on the right side, supposed to be neuralgia. He became dull, heavy, and stupid, comatose, and died. This man's gait was very unsteady. He said people must have thought him intoxicated.	Abscess in the right lobe of cerebellum, containing fetid pus. Ulceration of dura mater, over a carious portion of the temporal bone.	Cannot be calculated.	Recorded by Mr. Toynbee, p. 324.
73	Male	13	Caries of the petrous bone.	In June, 1851, suffered from so-called simple fever. He had severe headaches, chiefly in the right temporal region, and discharge from the right ear. Pain never entirely left. 12th, pain increased. His speech became thick and indistinct, at times almost unintelligible. No fever. 13th, he dragged his right leg. Vomited and became drowsy. 16th, vomiting and drowsiness continued. Had occasional double vision. 17th, decidedly improved. 19th, able to come down stairs. July 2nd, symptoms returned with greater violence than ever. Vomited frequently. Intolerance of light. Mind clear. July 7th, again improved. Pain, drowsiness, and sickness diminished. 13th, much improved. 15th, again he became worse. Severe paroxysms of pain, followed by stupor. He died in one of the paroxysms, July 17th.	The under surface of the right lobe of cerebellum attached to the dura mater. Three abscesses in this lobe. Two lined by distinct membrane. Almost the whole of this lobe was a bank of pus.	Cannot be calculated accurately.	Recorded by Mr. Toynbee, p. 328.

No.	*Sex.*	*Age.*	*Cause.*	*Symptoms.*	*Seat of Abscess.*
74	Male		Injury to the head. A splinter of wood had penetrated the skull. This occurred on Oct. 1, 1864; he died Oct. 27, 1864.	Suffered from the effects of the explosion at Erith, and received a penetrating wound of the skull by a piece of wood. A splinter had been driven in. It was removed, and becoming worse he was removed to Guy's Hospital. Mr. Cock made an incision. Some pus escaped, and after the pus some clear fluid followed. The child appeared relieved, and went to sleep, soon got worse, and died three days afterwards.	Autopsy showed an oval opening in the skull at the back part of the head on the right side. Acute arachnitis on both sides, more on the right. An abscess, the size of a walnut, in the back part of the right hemisphere, just below the descending cornua; purulent matter also in the lateral ventricle. The clear fluid that escaped was probably ventricular.
75	Female	31	Tubercular disease of the lungs, Fallopian tube, and uterus.	Two weeks before admission into Guy's Hospital she came one day as an out-patient, with a "wet rag" on the top of her head. She complained bitterly of the pain in her head. Her tongue was furred, and she had some pyrexia. Thinking it might be fever, she was ordered salines, and advised to come into the Hospital. About eleven days afterwards she came again, and "still had the rag on her head," and complained very much of pain in the head. This was her principal symptom.	Post-mortem examination showed no disease of the bones. A small tumour attached to the surface of the dura mater, imbedded into the edge of the hemisphere, near the longitudinal sinus. Vascular arachnoid. On surface of brain a thick cyst the size of a grape, and filled with liquid pus. In the brain substance there were three abscesses exactly like it. Each had a thick cyst, which was vascular, and could be easily turned out. All close to the surface. All the size of small chesnuts. Lungs filled with tubercular matter. Fallopian tube near its end filled with a soft tubercular and purulent matter; also the uterus, near the opening of the tube.
76	Male	35	Cause unknown. Had he had any blow on the head? The so-called fainting-fits' evidence tended to show some long-standing cerebral affection?	Led an irregular life. Employed at a music hall, and some years ago got his chest crushed in an accident, and said after the injury that he spat blood. Not known that he had injured his head. All that could be learnt of any previous illness was that he had been subject to "fainting fits." Said to have been well until three days before admission, when he was seized with tingling and numbness in the left hand; this increased, the arm got weak. On admission, much loss of power on the left side: this increased. A week before he died, complete hemiplegia. Twitching on the right side. Became insensible and died.	Post-mortem examination:—Calvarium was irregularly thickened in parts, as if there might have been an ostitis at some previous time, but no caries was discovered. Arachnoid slightly greasy and opaque. A large abscess in the left hemisphere, with a distinct wall, evidently some weeks old. It contained offensive viscid yellow pus. Had opened into the left lateral ventricle. Lungs congested. Heart, liver, kidneys, bladder, spleen, peritoneum, all healthy.

⁂ The last three cases are recorded in Guy's Hospital Post-mortem Records. Dr. Wilks has kindly allowed us to make use of them.

B. Partial Diseases of the Nervous System (*continued*).

2. Diseases of the Spinal Column.

Meningitis.
Myelitis.
Congestion.
Tetanus.
Locomotor Ataxy.
Irritation.
General Spinal Paralysis.
Hysterical Paraplegia.
Reflex Paraplegia.
Infantile Paralysis.
Hæmorrhage.
Non-inflammatory Softening.
Induration.
Atrophy and Hypertrophy.
Tumour, etc.
Concussion.
Compression.
Caries of Vertebræ.
Spina Bifida.

DISEASES OF THE SPINAL CORD.

By C. B. Radcliffe, M.D., F.R.C.P.

A. Preliminary Remarks.

Before proceeding to cope with the intricate and difficult pathological topics which form the subject of the present article, it appears to be expedient to glance at some points in the physiology of the spinal cord, and also to try and ascertain the true significance of pain, spasm, and certain symptoms analogous to pain and spasm, which figure conspicuously in the histories of spinal maladies; for if these matters be not disposed of as preliminaries now, they will prove to be the cause of frequent and distracting digression afterwards.

I. A Glance at some Points in the Physiology of the Spinal Cord.

1. *Roots of spinal nerves.*—The result of recent researches has been to establish in the fullest manner the truth of Sir Charles Bell's great discovery, that the posterior roots of the spinal nerves are devoted to sensation only, and the anterior roots to motion only. In one article, at least, the creed of to-day is the same as that of yesterday: and is some comfort to have it so, for in many other articles the creed of yesterday is not that of to-day.

2. *Posterior columns.*—If these columns be cut across, the result is, *not* numbness, as it would be if these columns were, as was once supposed, simply the continuation of the posterior roots of the spinal nerves, but hyperæsthesia and loss of co-ordinating power in the parts below the section, with a certain degree of local pain; and on inquiring further, it is found that this pain is due, not to any sensitiveness in the columns themselves, but to the irritation of the cut having travelled through the posterior roots of the spinal nerves, which posterior roots, as the researches of Lockhart Clarke show, pass through the posterior columns, more or less directly, to the central grey matter of the cord.

3. *Restiform bodies and small posterior pyramids.*—What has just been said of the posterior columns of the cord appears to apply equally

to the restiform bodies, and to the small posterior pyramids of the medulla oblongata, which pyramids lie between the restiform bodies posteriorly. Hyperæsthesia and incoordination in the parts below the section, with some local pain, are still the result of cutting these parts across: and as the connexions of these parts are above with the cerebellum, and below with the posterior columns of the cord, the natural inference is, that the channel through which the cerebellum acts upon the body is formed of the restiform bodies and small posterior pyramids in the upper part of its course, and of the posterior columns of the cord in the lower part.

4. *Anterior columns.*—If one of these columns be cut across, it ceases to act on that side of the body in the parts below the section, and the paralysed parts are benumbed to a certain degree, unless the cut be made in the part which lies immediately below the anterior pyramids of the medulla oblongata. In this part the anterior pyramid may be cut across without causing any very obvious paralysis or loss of sensation: in this part the results of dividing the anterior column at a lower level are only obtained when the cut is extended transversely, so as to divide the lateral column. It is plain, in fact, that in the uppermost part of their course the anterior columns have not that intimate connexion with the anterior roots of the spinal nerves, and that all-important part to play in voluntary movement, which they evidently have everywhere else: and it is also plain that the anterior columns, where they have to do with voluntary movement, have also something to do with sensation, for it is a fact that a certain degree of numbness is produced by the injuries which give rise to paralysis.

5. *Anterior pyramids.*—A transverse section of one of the anterior pyramids of the medulla oblongata in any part of its course annihilates all power of voluntary movement in the muscles below the section on the *opposite* side of the body, without affecting the sensation in any appreciable manner; and thus it is plain, not only that each pyramid contains very many, if not all, the conductors concerned in carrying the orders of the will to the muscles of the opposite side of the body, but also that the conductors which are collected in one pyramid decussate with those collected in the other pyramid at the lower and not at the upper boundaries of the pyramids. In a word, all the evidence, old and new, goes to show that these bodies are composed of conductors concerned in voluntary motion without any admixture of sensory conductors.

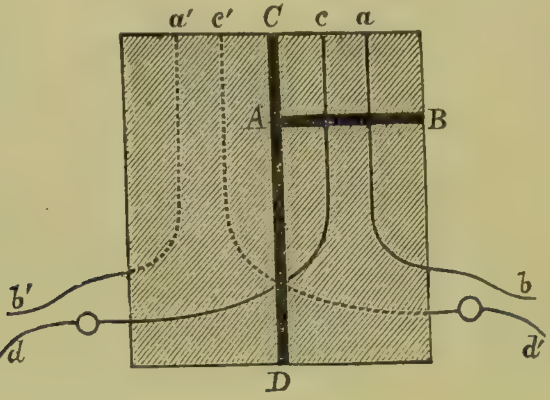
6. *Lateral columns.*—In the cervical region, for a short distance below the point at which the anterior pyramids of the medulla oblongata intercross, the lateral columns of the spinal cord have certainly very much to do in transmitting the orders of the will to the muscles; for, as has just been seen incidentally, the muscles below the section on the same side of the body are paralysed by cutting one of them across in this part. In the lower part of the cervical region, and in the dorsal and lumbar regions, it is very different, and the difference

is not very clearly determined. Here some trifling paralysis may be produced by dividing these columns transversely, but never more than this. Here, indeed, it would seem that this operation is followed by a certain degree of anæsthesia, and by the same result, as regards movement, as that which follows transverse division of the posterior columns—that is, not by paralysis, but by incoordination. A certain degree of anæsthesia appears to be a constant consequence of cutting across the lateral columns in any part of their course; and herein would seem to be an important distinction between the lateral and the posterior columns, for, as has been stated already, the result of cutting across the posterior columns is to produce hyperæsthesia, not anæsthesia.

7. *Olivary bodies.*—A section of the olivary bodies is followed, not by any marked degree of paralysis, or anæsthesia, but by a state of persistent spasm in many muscles on the *same* side of the body, in the neck especially,—a state which may some times continue for days, weeks, or even months. It is found, also, that this strange result is produced by irritating several parts of the base of the encephalon, the lateral and posterior parts of the medulla oblongata and pons varolii especially, as well as by irritating the olivary bodies. These parts are not very clearly defined. "They seem," says Dr. Brown-Séquard, "to be quite different from those employed in the transmission of sensitive impressions, or of the orders of the will to the muscles, at least in the medulla oblongata and pons Varolii. They constitute a very large portion of these two organs, and, perhaps, as much as three-fourths of the one first named. They are placed chiefly in the lateral and posterior columns of these organs; and because many of their fibres do not decussate, the spasm produced by irritating them is on the *same* side of the body."

8. *Grey substance of the cord.*—Instead of being merely a nerve-centre—the special centre of Marshall Hall's excito-motor system of nerves—there is now reason to believe, with Dr. Brown-Séquard, that the grey substance of the spinal cord is an important conductor of sensory and motor impressions. Paralysis without loss of sensation on the same side of the body, loss of sensation without paralysis on the other side of the body, are the strange results of cutting across one lateral half of the grey substance of the spinal cord: anæsthesia on both sides of the body, paralysis on neither side, are the equally strange results of making a longitudinal section midway between the two lateral halves: these are the two great facts which, when properly interpreted, furnish the reasons for believing, not only that there are sensorial and volitional conductors in the grey substance of the cord, but also that these two forms of conductors follow a different and definite course. Nor is it difficult to see how this may be. Let the course of the conductors in connexion with the anterior and posterior roots of a pair of spinal nerves be what is represented in the following diagram,—*a b* being the motor conductor descending to the right, and *a′ b′* the corresponding conductor descending to the left; *c d* being the

sensory conductor ascending from the left, and *c′ d′* the corresponding conductor ascending from the right,—and very little reflection will serve to supply the demonstration wanting. With the sensory and motor conductors arranged in this manner, it is plain that a cut across the right lateral half of the grey substance—a lesion indicated in the diagram by the line A B—must destroy the continuity of the motor conductor *a b*, and of the sensory conductor *c d*, and leave untouched the motor conductor *a′ b′*, and the sensory conductor *c′ d′*—must bring about, that is to say, what has been seen to happen in the first of the two experiments under consideration; namely, preservation of sensation with loss of motion on the side of the lesion, and preservation of motion with loss of sensation on the opposite side. Again, with the sensory and motor conductors arranged in this manner, it is plain that a section of the grey substance of the cord midway between the two lateral halves—a lesion indicated in the diagram by the line C A D—must leave the motor conductors *a b* and *a′ b′* untouched, and cut across the sensory conductors *c d* and *c′ d′* at their point of decussation—must bring about what happens in the second of these two experiments, viz. numbness on both sides of the body, and paralysis on neither side.



In saying that paralysis without loss of sensation, on the same side of the body, and loss of sensation without paralysis, on the other side of the body, is produced by cutting across a lateral half of the spinal cord, all is not said that has to be said. In such a case there is, in addition, increased temperature and sensibility on the side on which sensation is preserved, and diminished temperature on the side on which sensation is lost, especially if the section be made high up near the medulla oblongata. It would seem, in fact, that the injury has acted upon the vaso-motor nerves contained in the cord as well as upon the common motor and sensory nerves, causing paralysis of vaso-motor nerves on the side on which there is increased temperature and sensibility, and irritation of vaso-motor nerves on the side on which there is diminished temperature and anæsthesia. At any rate this mode of explanation is neither impossible nor improbable. The experiments of Professor Claude Bernard, Dr. Brown-Séquard, and others upon the cervical sympathetic, prove that when this nerve is paralysed by dividing it, a state of hyperæmia, of which the most conspicuous signs are a bloodshot state of the conjunctiva and of the lining membrane of the nostril and ear, with a contracted pupil, and with increased temperature, is at once set up on the same side of the head: and also that when the end of the divided nerve below the section is irritated, the immediate result is dilatation of the pupil, with an almost complete blanching and cooling of the parts which were bloodshot and warm a moment before. The vessels in these parts evidently relax and receive more blood when their nerves are paralysed, and contract and receive less blood where their nerves are irritated; and the increased temperature and sensibility which happens in the one case, and the diminished temperature and sensibility which happens in the other case, are nothing more than the natural consequences of the increased or diminished quantity of blood in the parts in each case respectively. All this is plain enough. Moreover, there are other facts which go to show that phenomena in every way analogous to those which result from paralysis or irritation of the cervical sympathetic are produced by paralysing or irritating vaso-motor nerves in other parts. There is, therefore, no reason why it may not be inferred that the increased temperature and sensibility of one side of the body, and the diminished temperature of the other side, which happen when a lateral half of the spinal cord is cut across, are the result of vaso-motor nerves being paralysed in the one case and irritated in the other case. Nay, such an assumption is well-nigh inevitable, for the structural connexion between the spinal and sympathetic systems of nerves is such as to make it scarcely possible to believe that a lateral half of the cord can be cut across without paralysing and irritating vaso-motor nerves.

9. *Motor and sensory tracks.*—The conductors concerned in voluntary motion, and those belonging to common sensation, both intercross in the cord, but not at the same place. From the *right* side of the brain, voluntary impressions pass to the motor nerves of the *left* side of the body, their course thither being, first, down the *right* anterior pyramid, then across to the *left* lateral column, then for a short distance down the *left* lateral column, then down the *left* anterior column, and to some extent also down the *left* side of the grey substance and the *left* lateral column, and so out at the *left* anterior roots: from the *left* side of the brain, these impressions pursue a similar course, only passing to the right side of the body instead of the left. Entering at the *right* posterior roots of the spinal nerves, the impressions which give rise to common sensations pass to the left side of the brain, up the *left* side of the grey substance, and to some extent also up the *left* lateral column and the *left* anterior column, the crossing to the other side of the cord being at the level of the entrance of the conductors into the cord, or thereabouts: entering at the *left* posterior roots of the spinal nerves, the impressions in question take a similar course to the *right* side. Both sets of conductors intercross in the cord, but

not at the same place. The conductors concerned in voluntary motion intercross at the decussation of the anterior pyramids. The intercrossing in this case is at this place, and at this place only: there is none above it, none below it. The conductors belonging to common sensation, on the other hand, intercross below the decussation of the anterior pyramids, and throughout the whole length of the cord. These are the main points to be remembered with reference to the tracks of these two forms of conductors in the cord.

The conductors which have to do with co-ordination of movement appear to be confined to the posterior columns of the cord, and to the parts which connect these columns with the cerebellum, the restiform bodies, and the small posterior pyramids. They are quite distinct from the conductors concerned in voluntary movement, and they also differ from these conductors in this, that those belonging to the two sides of the body do not intercross anywhere.

The vaso-motor conductors which enter into the composition of the cord appear to lie chiefly in the grey substance, for the dilatation of vessels resulting from paralysis of these nerves is brought about by dividing the grey substance rather than the white. Moreover, the fact that this dilatation of vessels is on the *same* side as that on which the grey substance is divided, must be taken as a reason for believing that the vaso-motor conductors belonging to the two sides of the body, like the conductors which have to do with the co-ordination of movement, do not intercross in the cord.

And so likewise with certain other conductors of a vaguer sort. These lie in and about the olivary bodies, and in the upper third of the lateral column; and there is, as it would seem, no intercrossing between those belonging to the two sides of the body, for the simple fact is this, that the persistent spasm which is brought about by irritation, which spasm is the only fact pointing to the existence of these conductors, is always on the same side as that to which the irritation is applied.

On the other hand, there appears to be nothing peculiar in the sensory conductors which are not concerned in common sensations—those which have to do with pain, tickling, temperature, and the rest. What has been said of the common sensory conductors would seem to apply to them in every respect, and indeed it may be doubted whether different conductors are required for the transmission of the different kinds of impressions.

10. *Increased reflex action.*—When the continuity of the cord is entirely interrupted by being cut, torn, compressed, or injured in any other way, voluntary movement and sensation are abolished in the parts below the injury; and at the same time the paralysed muscles, especially in the lower extremities, become much more prone to reflex action. This increased proneness to reflex action is developed immediately, or all but immediately, and it may continue with little or no change for days, weeks, or even months. It makes its appearance before there is time for the development of inflammation or congestion;

it continues after the time when any inflammation or congestion resulting from the injury which led to it may be supposed to have come to an end; and therefore it is difficult to look upon it as an indication of inflammation or congestion. Indeed the history of inflammation or congestion of the cord is opposed to this idea, for most certainly increased reflex action does not figure among the symptoms of unequivocal instances of these disorders. And this is all that need be said now, except this, that the history of increased reflex action would seem to be more intelligible on the view of muscular action which recommends itself to the writer, than on that which is commonly accepted.

11. *Increased temperature.*—In a paper on injuries of the spinal cord, published more than thirty years ago,[1] Sir Benjamin Brodie says:—"M. Chopat has given an account of some experiments on animals, in which he found that the division of the superior portion of the spinal cord produced a remarkable evolution of animal heat, so that it was raised much above the natural standard. I have made experiments similar to those of M. Chopat, and have met with similar results. I have also seen several cases in which an accidental injury of the spinal cord has produced the same effect. The most remarkable of them was that of a man who was admitted into St. George's Hospital, in whom there was a forcible separation of the fifth and sixth cervical vertebræ, attended with an effusion of blood within the theca vertebralis, and laceration of the lower part of the spinal cord. Respiration was performed by the diaphragm only, and of course in a very imperfect manner. The patient died at the end of twenty-two hours; and, for some time previously to his death, he breathed at very long intervals, the pulse being weak and the countenance livid. At last there were not more than five or six inspirations in a minute. Nevertheless, when the ball of a thermometer was placed between the scrotum and the thigh, the quicksilver rose to 111° of Fahrenheit's scale. Immediately after death the temperature was examined in the same manner, and found to be still the same." A Russian observer, Dr. Tscheschechin,[2] has also ascertained that considerable elevation of temperature, with quickened pulse and breathing, follows a section of the pons at its junction with the medulla oblongata, and that these symptoms go on increasing for two or three hours, until the state is that of high fever. Moreover, increase of temperature on one side of the body and decrease on the other has been seen to be one effect of dividing one-half of the grey substance of the cord. There is, indeed, reason to believe, not only that increased temperature is one effect of division of the cord, but that this change is in some way connected with paralysis of vaso-motor nerves; for in speaking previously of the experiment last mentioned, it was shown that this paralysis may well be supposed to lead to this result.

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[1] Med.-Chir. Trans. vol. xx. 1837.

[2] Reichert's and Du Bois-Reymond's Archiv f. Anat. u. Phys. 1866.

12. *Hints for determining the level of the injury in certain forms of spinal paralysis, &c.*—If the injury be at the upper limit of the sacral region of the cord, the muscles of the bladder and anus will be paralysed, and so will the muscles of the lower extremities, with the exception of those which are supplied by the anterior crural and obturator nerves (the psoas, iliacus, sartorius, pectineus, adductor longus, a. magnus, a. brevis, obturator externus, vastus externus, v. internus, rectus femoris, &c.), which nerves come off from the second, third, and fourth lumbar pairs of spinal nerves. If the injury be very low down in the sacral canal, the compressor urethræ and the accelerator urinæ, as well as the sphincter ani, will be paralysed, but not the muscles of the legs; for the nerves of the three muscles, specified by name, come off almost from the extreme end of the cord, and below those which go to form the great sciatic. When the injury to the cord is higher up in the cord, in addition to the loss of voluntary power in the lower extremities and in the bladder and anus, the respiratory muscles will be more or less paralysed. If the injury be at the upper limit of the lumbar region, the lateral muscular walls of the abdomen will be paralysed, and so will all the muscles of the lower extremities, and one effect of the paralysis of the abdominal walls will be to compromise greatly the expiratory movements of respiration. If the injury be high enough to paralyse intercostal muscles, inspiration will be interfered with as well as expiration, and the degree of interference will be in proportion to the number of intercostal muscles implicated. If the injury be low down in the cervical region, all the intercostals will be paralysed, and so will the muscles of the upper extremities, except those of the shoulders, which receive their nerves from higher portions of the cervical region. If the injury be at or above the middle of the cervical region—at or above the level of the fourth cervical pair of spinal nerves—death will at once result from the suspension of all inspiratory movements. In this latter case it is customary to ascribe the stoppage of breathing to paralysis of the nerve which supplies the diaphragm—that is, the phrenic; but this explanation does not go far enough. The injury which paralyses the diaphragm paralyses the scaleni, the inter-costales, and the serrati magni, which muscles elevate the ribs in ordinary respiration, and in so doing play a part which is scarcely less important than that played by the diaphragm; and not only so, but it paralyses also the greater number of those accessory respiratory muscles which, acting upon and from the shoulders, come to the rescue when a great effort at inspiration is necessary, and produce additional expansion in the upper part of the chest. Not only is there a great difference between calm respiration and forced respiration, but there is a great difference also between the respiration of males and that of females. "In males," says Dr. Hutchinson, "the abdomen first bulges outwards, and the ribs and sternum nearest to the abdomen quickly follow this movement, until the motion, like a wave, is lost over the thoracic region. In females, the breathing com-

mences with a gentle heaving of the upper part of the thorax, more or less apparent according to the fulness of the mammæ, and with some slight elevation of the shoulders; and this movement of expansion spreads from rib to rib in a downward direction, and any bulging of the abdomen from the descent of the diaphragm is distinctly after this heaving of the lateral walls of the chest, not before it." In females also this bulging of the abdomen is so inconsiderable that the number of respirations cannot be counted by the hand resting on that region as it can be in the male. In calm breathing, in fact, the diaphragm does more and the ribs do less in males than in females; and this difference is so real that, for the sake of distinction, calm breathing may be spoken of as diaphragmatic in males, and as costal in females. This difference is such, indeed, that respiratory movements which are healthy in women are morbid in men; and *vice versâ*, that movements which are healthy in men are morbid in women. "In forced breathing," Dr. Hutchinson again says, "the greatest enlargement of the thoracic cavity in both sexes is made by the ribs and not by the diaphragm, as is generally believed;" and that this statement expresses what really happens, appears to be evident in the fact that in such breathing the hollow at the pit of the stomach, instead of being filled out and protruded, as it must be if the diaphragm descended in any marked degree, is actually drawn in and depressed. In forced breathing, indeed, the costal inspiration of women becomes more costal, and the diaphragmatic inspiration of men changes from this form to the costal. It is certain, however, that there may be forced diaphragmatic breathing as well as forced costal breathing, and that the one may be made to take the place of the other by an easy effort of the will, or by changes of position which interfere with the action of the diaphragm on the one hand, or of the ribs on the other. There is, indeed, no difficulty in understanding why diseases which interfere with the action of the diaphragm or ribs should make the breathing costal or diaphragmatic, as the case may be. As regards the expiratory movements of respiration there is little to say. In tranquil breathing, in males and in females alike, expiration is performed by the relaxation of the diaphragm allowing the abdominal viscera to press up into the position from which they had been depressed in inspiration by the contraction of this muscle, by the relaxation of the costal muscles allowing the ribs to spring back into the position from which they had been pulled up in inspiration by the contraction of these muscles, and by the resiliency of the air-passages themselves. In forced expiration the lateral and inferior muscular walls of the abdomen will help to empty the chest by pulling down the ribs and by contracting upon the abdominal viscera, so as to cause them to push up the diaphragm more effectually. It is easy, indeed, to see how a lesion of the spinal cord which paralyses the lateral and inferior abdominal walls must interfere with the movements of expiration, and especially with such violent movements as coughing or sneezing. In a word, the whole case of the respiratory movements is one which makes it impossible to continue in the belief, that the one reason why the division of the cord at or above the origin of the phrenic nerve proves fatal, is because the diaphragm is paralysed; for the plain fact is, that the injury which paralyses the diaphragm paralyses the muscles which elevate the ribs, both ordinary and extraordinary, and so puts an end to movements which are quite as important as those of the diaphragm, if not more so, in carrying on respiration. Of the other phenomena which may be present when the injury which interrupts the continuity of the cord as a conductor is in the neck, but not so high as to destroy life immediately, and which are not likely to be present when the injury is much below the cervical region, difficulty of swallowing, difficulty in vocalization, contraction of pupils, palpitation, and priapism appear to be the most important.

II. ON THE PRACTICAL SIGNIFICANCE OF PAIN AND SPASM, AND OF CERTAIN OTHER SYMPTOMS MORE OR LESS AKIN TO PAIN AND SPASM.

Have these symptoms to do with inflammation, or with a state which, though not unfrequently passing into inflammation, is in reality diametrically opposed to inflammation? This is the question to which I propose now to seek the answer, first, in relation to pain and the symptoms akin to pain, and, secondly, in relation to spasm and the symptoms akin to spasm.

1. *On the practical significance of pain and the symptoms akin to pain.*—There are some points in the history of common neuralgia—the beginning and ending of the paroxysm periodically at a given time, the association of the pain with rigors, the frequent ending of the pain in an obscure fit of feverishness, and others—which are calculated to suggest some relationship between this disorder and ague. It would seem, indeed, especially in that form of neuralgia which is met with in agueish districts, as if the neuralgia and the rigors were companion symptoms—as if there was some connexion between the pain and a depressed state of the circulation such as is met with in the cold stage of ague. There is also some reason to believe that neuralgia is antagonised rather than favoured by inflammation and fever. It is no uncommon thing for the history of facial neuralgia or tic-douloureux to be this:—first, neuralgia, without local tenderness and swelling, and redness, and with frequent chills and shivers, and a decidedly depressed state of the circulation; afterwards, cessation of neuralgia, cessation of chills and shivers, with local tenderness, redness, and swelling, and with some slight feverish reaction. What I have experienced in my own person, as well as what I have witnessed in others, enables me to speak with all confidence upon this point. It is also the rule, rather than the exception, for the *neuralgic* pain of toothache to come to an end when the face becomes swollen and inflamed; and it does not seem to be otherwise with the stabbing

neuralgic pains which so generally precede the inflammatory eruption of herpes, for it is usual for these pains to subside concurrently with the development of the eruption. Nay, I know of several cases of sciatica, in which the relief to the neuralgic pain was coincident with the development of a tenderness which seemed to betoken neuritis at one or more points in the course of the painful nerve, and in which, after this change, the patient was comparatively free from pain so long as the lame limb was kept still and let alone. With respect to neuralgia, in all its manifold forms, indeed one thing is certain, and this is, not only that neuritis is not necessary to its production, but also that this form of inflammation is at most a very exceptional complication.

Nor is a different conclusion to be drawn from the history of rheumatic and gouty pain.

In acute rheumatism it is generally found that the pains which had been torturing the patient for days, or weeks, or months previously, preventing him from being at ease in the daytime, and causing him to toss about in sleepless misery at night, come to an end when the feverish reaction and local inflammation of the fully-formed disorder make their appearance. After this, the joints are *tender* enough; but if the patient keep as still as he is very likely to do under the circumstances, he is comparatively or actually at ease so far as his old rheumatic pains are concerned. Or, if it be otherwise, the pains will generally be found to be in a part in which the signs of rheumatic inflammation are imperfectly established or absent, or else at a time when there is a decided remission in the feverish reaction—an event which happens more frequently in this disorder than is commonly supposed.

And certainly it is impossible to look upon the local inflammation of gout as essential to the racking pain of this disorder. "About two o'clock in the morning," says Sydenham, who knew full well from personal experience what he ought to say, "the patient is awakened by a severe pain in the great toe, or, more rarely, in the heel, ankle, or instep. The pain is like that of dislocation, and yet the parts feel as if cold water were being poured over them. Then follow chills and shiverings, and a little fever. The pain, which was at first moderate, becomes more intense; and with its intensity the chills and shivers increase." After tossing about in agony for four or five hours, often till near daybreak, the patient suddenly finds relief, and falls asleep. Before falling asleep, the only visible change in the tortured part is some swelling in the veins; on waking in the morning the part has become swollen, shining, red, tender beyond measure, and more or less painful, but painful only to a degree which is as nothing in comparison with the torture of the night past. It seems, indeed, as if the pain which now exists may in great measure be referred to the mere tension and stretching of the inflamed ligaments, for it may be relieved, or even removed, by judiciously applying support to the toe and sole of the foot. On the night following, and not unfrequently for the next three or four nights, the sharp pain may return, reappearing and disappearing suddenly, or almost suddenly, and resulting in the development of additional inflammatory swelling in the interval between falling asleep and waking in the morning. The pain in these relapses, like the pain in the first attack, is accompanied by chills and shivers, and by the most distressing irritability and excitability; but, until unequivocal signs of inflammation are developed in it, the painful part is not tender in the true sense of the word. The inflammation, moreover, is attended by no fever, or by very little; or if it be otherwise, as it is occasionally, the inflammation runs higher than usual, *and the characteristic pain is less urgent than usual.* Dr. Garrod points out this latter fact in his excellent work on Gout. From its history, then, it would seem as if the pain went hand in hand with the rigors which belong to the cold stage of gouty inflammation. It would seem as if the inflammation, as inflammation, had little to do with the pain; for if it were otherwise, it is scarcely to be supposed that the pain should be least urgent in the cases of gout in which the inflammation is most marked, and that the unequivocal signs of inflammation should make their appearance during sleep without waking the patient. Nay, it would even seem as if the pain were put an end to by the establishment of inflammation—as if, in fact, the pains were antagonised rather than favoured by the inflammatory condition. Moreover, the suddenness with which it begins and ends in the majority of cases must be looked upon as a reason for referring the pain to the category of neuralgia—a disorder with which, as I have already shown, inflammation has no necessary connexion.

There is also reason to believe that pain holds the same relation to fever and inflammation in other kinds of fever besides the rheumatic, and in other kinds of inflammation besides the gouty.

The pain in the back, often very severe, which ushers in small-pox, disappears before the hot stage is fully established. It comes and goes hand in hand with the rigors, and it belongs to the cold stage as evidently as do the rigors. And this would seem to be the case also in other fevers; for it is the rule, and not the exception, for the pains which attend upon the onset of these disorders to pass away or to become greatly mitigated as soon as the cold stage gives place to the hot. Nay, it would seem as if pain gave place for the time to what may be called artificial feverishness. At any rate, I have more than once felt tic-douloureux in my face pass away as soon as I could set my blood in brisk motion by violent bodily exercise; and on two occasions I have put a stop to a sudden attack of lumbago while in the saddle, by a practice which is not unfrequently adopted in such a case in the hunting-field,—that is, by leaning forwards, and beating the loins with the hands until the whole body was aglow, and the perspiration dropped from the forehead.

The acute pain of a dislocation or sprain—the pain to which

Sydenham likens that of gout—does not, as a rule, remain after the parts have begun to be hot and tender and swollen ; and as a rule, also, the pain of idiopathic inflammation goes before, and not along with, the redness and heat and swelling. In the idiopathic, as well as in the traumatic forms of inflammation, it would seem, indeed, as if the pain were related to the cold stage of the disorder, and not to the hot. Nor is a contrary conclusion to be drawn from the history of those cases in which the pain continues after the hot stage of the inflammation is fully established, for in these cases this persistent pain is evidently (in great measure at least) due to the stretching of parts made tender by the inflammation. Thus, for example, the pain which remains after the hot stage is fully established in orchitis and pleuritis, is at once removed or relieved by means which obviate this stretching,—in the former case by the free use of the knife, in the latter case by the application of a roller around the chest so as to prevent the movement of the ribs over the seat of inflammation.

Even in inflammation of the membranes of the brain, severe pain in the head cannot be looked upon as a symptom of this inflammation. About six years ago I had a youth in the Westminster Hospital with well-marked symptoms of acute cerebral meningitis. When I first saw him, he complained of frequent rigors and of a constant agonizing pain in the head, and at this time his face was pale and perspiring, his ears and his head generally were below the natural temperature, his pupils somewhat dilated, and his pulse contracted and feeble. Eight hours afterwards, when I saw him the second time, his face was flushed, his head burning hot, his pupils contracted, his eyes ferrety, his skin hot and dry, his pulse strong and full, and fierce delirium had taken the place of the pain. And this, so far as my experience goes, is the regular history of pain in this disorder. It is pain ceasing, not pain beginning, as the symptoms of active determination of blood to the brain make their appearance. It is pain in association with an anæmic rather than with a hyperæmic condition.

For these among many reasons it is that pain (with the exception of that form of pain which is dependent on tenderness, and which is accidental only) does not appear to be a symptom of inflammation or fever. In inflammation or fever the pain would seem to be connected with the cold stage preceding the hot stage, and not with the hot stage itself—with a state of capillary contraction and deficiency of blood, and not with a state of capillary relaxation and excess of blood—with a state of vaso-motor irritation, and not with a state of vaso-motor paralysis : in other cases, the pain would seem to have to do with a state of circulation which is in reality closely akin to that which exists in the cold stage of inflammation and fever. Pain, however, must not be regarded as a symptom of inflammation or fever because it happens to be associated with the so-called cold stage of these disorders. In point of fact, this so-called cold stage of inflammation or fever is a state which is diametrically opposed to the so-called hot stage. In this cold stage, the vaso-motor nerves (and not these nerves only) are in a state of irritation, and, as the result of this irritation, the capillaries are contracted and comparatively bloodless; in the hot stage, on the contrary, the vaso-motor nerves are paralysed, and, as the result of this paralysis, the capillaries are relaxed and bloodshot. Instead of being stages in the same process, the so-called cold stage and the so-called hot stage are conditions diametrically opposed to each other. Instead of being stages in the same process, it would rather seem that the hot stage has a remedial relation to the cold stage—that, within certain limits, the hot stage is the salutary refluence of a tide of life which has ebbed too low in the cold stage. It is not difficult to see that there is an intimate connexion between the so-called cold stage and the so-called hot stage, and that the first may easily change into the second. It is not difficult to see that there must be this relation between these stages; for if, as there is good reason to believe, irritation of vaso-motor nerves may bring about the cold stage by causing contraction of vessels, it is easy to understand that the paralysis of vaso-motor nerves, which follows when this irritation is carried beyond a certain point, may lead to the hot stage by causing relaxation of vessels. At any rate, be this as it may, the plain fact would seem to be that pain, with the exception of that form of pain which is dependent on tenderness, is a symptom belonging to the so-called cold stage of inflammation and fever, or to a state of circulation closely akin to it, and not to the hot stage of inflammation and fever, or to a state of circulation akin to it. Nay, it may even be supposed, and not without some show of reason, that pain *must* be associated with contracted and empty capillaries; for, the sympathies of the nervous system being what they are, it is not easy to believe that the vaso-motor nerves do not participate in the irritation of the sensory nerves, for which pain is the expression in words.

And if this be so—and this is the practical conclusion to which these remarks tend—it follows that pain is likely to be relieved by measures which are calculated to rouse the circulation and increase the quantity of blood in the capillaries of the painful part, and not by those which have a contrary action.

With regard to tingling and other symptoms which are more or less akin to pain, there is little to say. Indeed, all I can say is that the history of these symptoms, so far as it is known to me, would seem to agree rather than to disagree with that of pain, in connecting them with a state of irritation, and not with a state of actual inflammation.

2. *Of the significance of spasm and the symptoms akin to spasm.*—The violent and general epileptic form of convulsion which attends upon death by hæmorrhage or suffocation is associated with a defective and not with an excessive supply of arterial blood to one or other of the great nerve-centres. Nor is it otherwise with ordinary epileptic or epileptiform convulsion. The deathly paleness of the countenance which precedes the convulsion is, indeed, a plain proof that the fit

commences in a state of circulation which is the very opposite to that of active determination of blood to the head, and the strong pulse which is usually perceptible in the arteries as the fit progresses is no contradiction to this conclusion. This strong pulse is usually regarded as a sign of arterial excitement—as a proof that more arterial blood is being injected into the arteries at this time, and that, on this account, certain nervous centres are excited to an unwonted degree of activity: but the simple fact is, that the strong pulse which is present under these circumstances derives its strength, not from arterial blood, but from venous. Black blood is being pumped into the arteries at the time; and because black blood moves less readily through the capillaries than red blood, the arteries become distended and the pulse endowed with a counterfeit power. The strong pulse in question is caused by the suffocation which is a part of the fit: it is a pulse of black blood and not of red, as may easily be proved by making an opening into the artery: it is nothing more, in fact, than the natural pulse of suffocation. Hence, the strong pulse of the epileptic or epileptiform paroxysm is no proof that this form of convulsion is connected with an excited condition of the circulation: on the contrary, when rightly read, it points only to the opposite conclusion.

It would seem also that convulsion is not associated with an over-active condition of the circulation, even in those cases in which at first sight it might appear to be so. In the fevers of infancy and early childhood, especially in the exanthematous forms of these disorders, convulsion not unfrequently takes the place occupied by rigor in the fevers of youth and riper years. It occurs in the initial cold stage, or else in the last moments of life, not in the intermediate hot stage. Again in inflammation of the membranes of the brain, convulsion, when it occurs, is connected with the cold stage before the hot stage, or with the cold stage after the hot stage, and never with the hot stage itself. Nay, I am disposed to think that there is something altogether uncongenial between convulsion and a state of febrile reaction in the circulation, for it is a fact not unfrequently verified that fits of common epilepsy are often suspended during the continuance of such reaction.

As indeed I have endeavoured to show at length elsewhere,[1] the physiology and pathology of muscular action, so far as I can read them, serve only to connect all the varied forms of tremor, convulsion, and spasm, with diminished and not with increased activity of the circulation; and thus the practical significance of spasm and the symptoms akin to spasm would appear to be the same as that of pain and the symptoms akin to pain—namely this, that the measures calculated to afford relief are likely to be those which will rouse the circulation to greater activity and increase the quantity of blood in the capillaries, and not those which have a contrary action.

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[1] Epilepsy, &c.: Lectures delivered at the Royal Coll. of Phys. in London. Post 8vo. Churchill, 1862.—"Dynamics of Nerve and Muscle," and "Electrophysiologica," in "Nature," Jan. 4, 11, and 16, 1872. Post 8vo. Macmillan & Co. 1872.

B. On Diseases of the Spinal Cord.

Under the head of diseases of the spinal cord there is no lack of subjects. As of primary importance may be mentioned spinal meningitis, myelitis, spinal congestion, tetanus, locomotor ataxy, and spinal irritation; as of secondary importance, reflex paraplegia, infantile paralysis, hysterical paralysis, hæmorrhage, white softening, induration, atrophy, hypertrophy, tumour, concussion, compression, vertebral caries, spina bifida, &c. I shall take each of these subjects in the order in which it has been enumerated, and, as far as I can, apportion the limited space at my command (very limited for such a purpose) so that there may be room for saying most where most is wanted.

I. Spinal Meningitis.

Inflammation of the membranes of the spinal cord is usually associated with inflammation of the substance of the cord (myelitis) or with inflammation of the membranes of the brain, but uncomplicated cases do occur now and then, and with care it is not difficult to discriminate between the symptoms which are essential to spinal meningitis and those which are only accidental.

1. Symptoms.—In order to arrive at a knowledge of the symptoms of spinal meningitis, I will relate as a text one of five cases verified by post-mortem examination which have come under my own notice, and then proceed to see wherein it agrees with or differs from other cases of the kind. I choose an acute case rather than a chronic one, for it is only in the acute form of the disease that the symptoms are to be defined with certainty.

Case.—A lightly-made, delicate-looking youth, nineteen years of age, a cigar-maker by trade, was admitted into one of my wards in the Westminster Hospital on the 27th December, 1864.

(*a*) When I saw him first—this was on the day after his admission —he complained chiefly of pain in the back and great general weakness and weariness, and expressed his belief that he had got rheumatic fever. He was then sitting by the fireside, and looking very ill. On telling him that he had better lie down, he got up and walked towards his bed, or rather he attempted to do so, for the first step brought on a severe pain in the back and legs, with a feeling of faintness and want of breath, and he would have fallen if assistance had not been at hand. Very soon after lying down he passed about a quart of water without any difficulty.

(*b*) The account he gives of himself is this. A week ago, after being very tired by a long walk, he was seized by shiverings and sharp pain between the shoulders. During the next three days he was feverish and without appetite, but still able to go about and do his work. All this while, he had very little pain, and his nights were not disturbed. On the night of the fourth day from the commencement of the illness he was awakened by violent pain along the whole course of the spine, in the groins, and in the right leg. Next day the pain occurred several times in paroxysms, and was accompanied by a good deal of starting and jerking in the legs; and so also on the two days following. On the day before admission to the hospital, some difficulty in opening the jaw was experienced, and the paroxysms of pain, and jerking, and starting had become more frequent and urgent. All this while the bowels and bladder acted properly.

Dec. 28.—There is no material change since yesterday—not for the worse, certainly.

Dec. 29.—Last night, after three or four hours' sleep, the patient awoke with very severe pain along the spine and down both legs, and since that time the pain has recurred several times. These attacks are separated by intervals of comparative or complete ease, and instead of the jerks and starts, which went hand in hand with it previously, the pain is now accompanied by stiffness in the muscles of the back and legs. At the present moment (about 2 P.M.) the head is drawn back on the pillow, and considerable pain and stiffness in the neck is caused by moving it. Before making such movement the patient was free from pain and stiffness in this region. Asking him to try and sit up, he attempted to do so, but was stopped at once by a severe paroxysm of pain along the whole length of the spine and down the legs, and by the muscles in the painful parts becoming stiff. The action of the muscles produced in this way arched the body backwards almost as much as in ordinary cases of tetanus, and at the same time pursed up the mouth and eyes, and gave a set expression to the features generally, so that the patient for the time had the appearance of a person considerably older tahn himself. The pain went off in a few minutes, and soon afterwards the stiffened muscles relaxed. The effort to move one of the legs spontaneously gave rise to a sharp pain in the thigh and loins, and the limb became somewhat stiffened in a semi-flexed position, and this state of things did not pass off for several minutes: and passive movement produced the same result. There was no numbness: on the contrary, the condition of the skin as to sensation everywhere, as judged by pricking and pinching and by differences of temperature, was plainly that of slight over-sensitiveness. Pressure along the spinal column failed to detect tenderness anywhere, and the result of applying a sponge wrung out of hot water was equally negative.

In the course of the examination it was evident that any movement of the body, or neck, or legs, active or passive, gave rise to pain and stiffness in the muscles moved; and also that there was little or no pain or stiffness so long as the patient kept quite still. It was evident, in fact, that the muscles were relaxed, except perhaps in the neck, in the intervals between the paroxysms. The poor sufferer was evidently in a great strait, dreading all movement, because he knew full well what the effect of movement would be, and at the same time continually prompted by an intolerable feeling of unrest and fidgetiness to wish to have his position changed in a way which he could not or dared not compass by his own efforts: and it is difficult to avoid the conclusion that the stiffness is, in the main, an instinctive act to prevent the movement which gives rise to the pain, rather than spasm like that which is met with in tetanus. The arms are affected as well as the legs, but not to the same degree. They are weak—so weak that it is not easy to find strength to carry the food to the mouth, the left arm being somewhat the weaker of the two. The left arm also cannot be moved, either actively or passively, without giving rise to pain and rigidity, to pain shooting up between the shoulders, to rigidity flexing the limb somewhat at the elbow, and bending the thumb slightly into the palm: not so the right arm. There is no numbness in either arm, and no very decided over-sensitiveness. Mastication is difficult, and deglutition still more so, apparently from the muscles set in movement becoming stiff in moving. The breathing is shallow and slow; the pulse quick (130) and very wanting in strength; the skin profusely perspiring after a paroxysm, and hot and moist at other times. Thirst is much complained of. The bladder is full, and it cannot now be emptied voluntarily. The urine is acid. The penis is flaccid, and has been so ever since the commencement of the illness. The bowels have not acted. The pupils are equal and natural, and there is no headache or other "head symptom."

Dec. 30.—A tolerably good night has been passed, and this afternoon the patient thinks himself a little better.

Dec. 31.—There has been a bad night, and much ground has evidently been lost since the last visit. In a paroxysm which is just over, want of breath was experienced rather than pain. Sensation is still somewhat exaggerated everywhere. Urine cannot be passed without the catheter, but the bowels have responded to-day to a dose of castor-oil and spirits of turpentine which was administered yesterday. During my visit I had an opportunity of seeing the patient *after* a paroxysm as well as in it, and I quite satisfied myself that the muscular stiffness of the paroxysm soon passed off, and that in the interval between the paroxysms the muscles were relaxed, except perhaps at the back of the neck—with this possible exception, because all along the head remained drawn back to some degree upon the pillow.

Jan. 1, 1865.—The night has been perfectly sleepless, with now and then some trifling lightheadedness. The paroxysms of pain, stiffness, and difficulty of breathing are not so frequent (three hours have passed since the last), but the respiration is certainly shallower and less sufficient, and the pulse more rapid and unsteady. There is the same want of power over the bladder. When I left the ward, it was plain enough that the patient was sinking: when I returned two hours later all was over, death having happened in a fit of choking and suffocation caused by attempting to swallow a spoonful of beef-tea with a morsel of bread sopped in it. *In the agony, the patient not only sat up in bed, but got out of bed and stood for a moment with his hands bearing upon the shoulders of the nurse who had been* *feeding him.* The body was carefully examined after death by Dr. Bazire, and the following notes were taken at the time from his dictation:—

"Time, twenty-four hours after death. Weather frosty. Cadaveric rigidity well marked. The muscles of the back dark and highly congested. On cutting through the posterior arches of the vertebræ the vertebral vessels are seen to be gorged with dark fluid blood. There is no effusion of blood outside the meninges in the interior of the canal. The meninges are highly congested throughout the whole length of the canal, but to a considerably greater degree in the region between the scapulæ. In this latter region, in addition to the thickening, opacity, and intense red colour of the dura mater elsewhere, there are streaks in its substance of black coagulated blood. The arachnoid is intensely red, and the pia mater extremely congested in the same region. Beyond it, the dark red colour of the dura mater gradually passes into a lighter shade, and becomes a bright pink near the cauda equina in one direction, and near the medulla oblongata in the other. The arachnoid is whitish again near the cauda equina. There is no effusion of serosity, blood, or pus, either between the meninges or on the surface of the cord; indeed, there seems to be a smaller quantity than usual of cerebro-spinal fluid. The substance of the cord itself looks normal in consistence, colour, and size. The central vessel of the cord is highly congested, and on section of the cord there exudes from the centre fluid black blood in minute drops. The cerebral meninges are normal. The cerebral sinuses are highly congested, and the same appearances of congestion (due probably to the mode of death) are met with in the substance of the brain. The organ itself is normal."

The symptoms of acute spinal meningitis are plainly exhibited in this case, and there need be no difficulty in distinguishing those which are of primary importance from those which are secondary.

As symptoms of primary importance may be enumerated these:—fits of pain produced by movement along the spine and in the extremities; fits of muscular stiffness in the painful parts along with the pain; intervals of comparative or complete freedom from pain and muscular stiffness so long as movement can be avoided; absence of paralysis; some exaltation of sensibility; loss of power over the bladder; partial loss of power over the bowel; absence of spinal tenderness.

Fits of pain along the spine and in the extremities, produced by movement.—This pain, as I think, must be regarded as the most prominent symptom in acute spinal meningitis. It may be confined to the region of the spine, but more generally it shoots into the extremities, into the legs especially. As a rule, it does not shoot beltwise round the trunk. It is brought on by any movement of the trunk, and, in great measure at least, it may be prevented by avoiding such movement. It is often brought on also by moving one of the extremities, the pain in this case beginning in the limb, and extending thence to the spine. It seems to depend, in part at least, upon the same cause as the pain of pleurisy, viz. the dragging of an inflamed and therefore exquisitely tender serous membrane, and its character is certainly more like the pain of pleurisy than like that of rheumatism (to which latter it has been likened), for it occurs in the same sharp, sudden, breath-stopping catches.

Fits of muscular stiffness in the painful parts along with the pain.—It is usual to regard this stiffness as analogous to the spasm of tetanus: it is necessary, as I believe, to look upon it as expressing an instinctive act of muscular contraction, of which the object is to prevent pain by arresting certain movements which produce pain. The spine and extremities cannot be moved without causing pain: the stiffness prevents the pain by preventing the movement; this would appear to be the true view. This explanation, originally given by M. Dance as applying to the muscular stiffness in a case of acute spinal meningitis observed by him and recorded by M. Ollivier, applies perfectly to the muscular stiffness of the case which has been related as the text, and it applies, as I believe, with the same exactness to all cases of the kind. Indeed, I believe there can be no greater mistake than to confound the stifness in question with the spasm of tetanus. This will be seen more particularly when speaking of tetanus: and here I will only say that tetanus in its most violent form is constantly present where there are no signs of spinal meningitis, and that, in the few cases in which such signs chance to be met with, it may be supposed that the inflammation is a consequence rather than a cause of the irritation which gives rise to the tetanic spasm—a consequence of the irritation in the vaso-motor nerves having proceeded until it has issued in paralysis of the vaso-motor nerves. Nay, after what has been said in the preliminary remarks, it is not impossible that the spinal meningitis which is occasionally associated with tetanus may have served to counteract the spasm rather than to cause it. At any rate, it is certain that spasm of the spinal muscles is not so marked a phenomenon in acute spinal meningitis as in tetanus, and that it is not to be regarded "comme indiquant positivement la phlegmasie des membranes de la moelle;" and it is, to say the least, highly probable that the muscular stiffness which simulates true tetanic spasm is in great measure an instinctive act of muscular contraction to prevent a movement which produces pain.

Intervals of complete or comparative freedom from pain and muscular stiffness so long as movement can be avoided.—These intervals are sometimes of considerable length, even for days. According to my own experience, indeed, the rule would seem to be that as long as the patient can keep still, so long is he, comparatively at least, free from pain and stiffness—a rule which is very different from that which obtains in tetanus.

Absence of paralysis.—The patient is weak, very weak, and he seems to be paralysed, but in reality he fears to move because movement brings back the pain. "Les mouvements, qui sont en quelque sorte enchaînés par la douleur, ont moins de force, mais ils ne sont point paralysés." (Ollivier, p. 595.) Let this fear be forgotten, and it is possible not only to sit up, but to get out of bed and stand, as happened in the final agony of the patient whose case I have given. This power of movement has been noticed in several cases, of which one is related by Ollivier, and another referred to; and I believe it would be witnessed in all cases of *uncomplicated* acute spinal meningitis in which the fear of suffering pain from movement was not the one absorbing feeling.

Some exaltation of sensibility.—In the case which I have given there was some exaltation of sensibility as to touch, pain, and differences of temperature, but to no very marked degree; and this would appear to be the rule in cases of the kind. It would seem, indeed, that numbness is a purely accidental symptom, which is never present unless the substance of the cord is implicated in the meningeal inflammation.

Loss of power over the bladder.—In acute spinal meningitis, when the symptoms are fully developed, this particular symptom is scarcely ever absent, if ever. Before this time it may be absent, as it was in the case on which I am commenting; but this absence must certainly be looked upon as the exception rather than the rule. Not unfrequently the inability to empty the bladder is preceded by a state of irritability which makes it necessary to pass water almost incessantly.

Partial loss of power over the bowel.—On this point M. Ollivier makes a remark which is certainly true: "Je ferai remarquer que l'abolition des fonctions de la vessie persiste toujours au même degré depuis le commencement jusqu'à la fin, tandis qu'il n'en est pas de même pour l'intestin, puisqu'il y a assez souvent des garderobes naturelles dans les derniers temps de la maladie." (Vol. ii. p. 601.)

Absence of spinal tenderness.—This absence is certainly a common, if not a constant, feature of acute spinal meningitis. In some chronic cases, no doubt, there may be some local spinal tenderness, but on inquiry these prove to be cases in which the phenomena of spinal irritation are mixed up with those of spinal inflammation—in which the inflammatory affection is complicated with that condition of which, as will appear in due time, local spinal tenderness is the distinctive feature.

These are the points which may be regarded as of primary importance in comparison with those which have still to be considered, namely—absence of marked spasmodic symptoms, difficulty of mastication and deglutition, difficulty of breathing, no increased reflex excitability, no priapism, fits of perspiration, no active inflammatory fever, no marked "head-symptoms."

Absence of marked spasmodic symptoms.—The rigidity which attends upon the paroxysms of pain has been seen to be in the main an instinctive act of muscular contraction to prevent a movement which produces pain, and there appear to be no other symptoms of a spasmodic character which occupy a conspicuous place in the history of spinal meningitis. Or if there be any such symptoms, these are in all probability confined, as were the jerks and starts in the case under consideration, to that early period of the disorder in which it may be supposed that actual meningeal inflammation was not developed—to the so-called cold stage of the disorder probably.

Difficulty of mastication and deglutition.—This difficulty is often absent, and when present it is at most a trifling trouble comparatively. There is no true trismus as in tetanus; there is at most only stiffness which prevents the jaws from opening easily and moving freely. This stiffness, moreover, is late in making its appearance, whereas in tetanus trismus is one of the very first symptoms. In a word, difficulty of mastication and swallowing would seem to occur only in those cases of spinal meningitis in which the higher portions of the cord are implicated.

Difficulty of breathing.—This difficulty is always present in some degree, and especially during a paroxysm of pain and stiffness. In some cases, indeed, the movement of the chest may be actually suspended at this latter time, and death may happen from this cause, as indeed was the case in a patient whom I saw not long ago with Dr. Julius of Richmond.

No increased reflex excitability.—This is not, perhaps, what might be expected theoretically: but, be the explanation what it may, the fact would seem to be that reflex irritability is not increased in acute spinal meningitis in the way in which it is ordinarily increased in tetanus. So far as I have been able to ascertain, there would seem to be no material change of reflex excitability in the meningeal inflammation.

No priapism.—The cases in which erection of the penis would seem to be a symptom appear to be those in which the substance of the cord is affected rather than the membrane—cases, too, in which the seat of the disease is in the cervical and upper dorsal region rather than in the lumbar region. At any rate, it would seem to be the rule for the penis to be flaccid in uncomplicated cases of acute spinal meningitis.

Fits of perspiration.—As in tetanus these follow a paroxysm almost invariably, especially in the latter stages of its disease. Of this there appears to be sufficient evidence.

No active inflammatory fever.—Thirst is a frequent symptom throughout, and there may be at first some heat of skin, but in the most acute cases there is little or no active sympathetic fever. On the contrary, there is usually, even in the cases which have most claim to be considered as acute, a decided want of febrile reaction from the beginning to the end.

No marked head-symptoms.—In very many cases inflammation of the spinal meninges is only a part of a more general disorder in which the cerebral meninges are also implicated, and, therefore, "head-symptoms" of one kind or other will often enough be mixed up with the spinal symptoms; but in cases like the one under consideration, where the spinal meninges were alone inflamed, "head-symptoms" do not figure at all, or figure only as phenomena of very secondary importance. Upon this point there is no lack of evidence. Where spinal meningitis is chronic in its course, its symptoms are often so mixed up with the Protean symptoms of spinal irritation (of which more in due time) as only to be detected with great difficulty. It may be suspected that the meninges are affected by inflammation rather than by simple irritation if fits of pain and stiffness are produced by movement in the spine and extremities, and if there be at the same time no spinal tenderness, no paralysis, and no tingling or numbness; and this is all that can be said except this, that this suspicion will gather strength if there be chronic disease in the bones and ligaments of the spine. But it may be questioned whether long-continued contraction of the muscles of one or more of the extremities or of the cervical muscles can be reckoned among these symptoms, for such contraction is certainly common enough in cases where the only condition of disorder in the spinal cord or its membranes is one which, from the sudden way in which it begins and ends, and for other reasons as well, would seem to be one of simple irritation.

2. POST-MORTEM APPEARANCES.—As Ollivier pointed out, the traces of spinal meningitis after death are met with usually, not in the arachnoid membrane, which is non-vascular, but in the subjacent vascular tissue. The arachnoid is so thin and transparent as to allow the vascular injection produced by the inflammation in the deeper structures to appear through it, and that is all. This injection is generally less evident on the surface of the cord than on that of the dura mater, because in the former place it is hidden by the effusion of turbid, sero-purulent, or purulent fluid in the space between the arachnoid and pia mater—in the space naturally occupied by the rachidian fluid—is hidden by an effusion which, before the arachnoid is opened, often causes the cord to have a swollen, opaque, yellowish-white, or yellowish appearance. Any fluid effusion is usually in this space, but sometimes there may be fluid, in this case often sanguinolent, in the space outside the dura mater, especially if there be disease in the bones or ligaments of the spine. Sometimes the rachidian space is obliterated here and there by inflammatory adhesions; sometimes the surface of the arachnoid is roughened or otherwise altered by calcareous or other deposits in patches; sometimes the opposed surfaces of the arachnoid are more or less adherent: but generally these surfaces are smooth and free, and the inflammatory products are met with below this membrane, and not above it. Very often, also, the proper signs of spinal meningitis are mixed up with those of cerebral meningitis or myelitis, or with those of disease in the bones or ligaments of the spine.

3. CAUSES.—The causes of spinal meningitis are often very obscure. In some cases it is rheumatism, or syphilis, or the suppression of some menstrual, hæmorrhoidal, or other habitual discharge, or the spreading

of cerebral meningitis downwards, or of disease in the bones and ligaments of the spine inwards, which would seem to figure as a cause; in other cases it is a casual injury to the back, or a chill caught by lying on the back on the cold and damp ground, or some particular disease, as tetanus, chorea, or hydrophobia, to which blame appears to belong. In fact, the causes are legion, and it is impossible to connect spinal meningitis with any particular cause or set of causes.

4. Diagnosis.—One or two points of diagnosis have been mentioned incidentally when dealing with the symptoms of spinal meningitis, and with these it is best to be content at present, for before this matter can be gone into advantageously materials must be had which can only be forthcoming when the phenomena of myelitis, spinal congestion, tetanus and other spinal maladies have been passed in review.

5. Prognosis.—Acute spinal meningitis is, without doubt, a very formidable and fatal disease. There are, indeed, few well-authenticated instances of recovery on record, and by some it is doubted whether there be any. Life may be cut short in four or five days, or it may be prolonged to twenty or thirty days, but not often—not often indeed—beyond six or seven days. In the sub-acute and chronic forms of the disease, the prognosis is of course less gloomy, but even here it is far from cheering.

6. Treatment.—In all cases of spinal meningitis, rest in the recumbent position, more or less strictly enforced according to the urgency or leniency of the symptoms, is indispensable, the best position, perhaps, being not strictly on the back, but rather upon the side, and with the limbs a little lower than the back, so as to favour the draining away of blood from the congested parts, and, at the same time, to facilitate the use of the local applications to the spine which may be necessary. Upon this point there can be little or no difference of opinion; upon all other points, in all probability, few will think alike. For my own part, I should be disposed to place most confidence in iodide of potassium and opium, with the local application of ice to the back in acute cases, and to bichloride of mercury, with counter-irritation in one form or other to the spine, in chronic cases. At the same time, I am inclined to think that the present fashion has set very unwarrantably against the old practice of giving calomel and opium, so as to affect the gums slightly and speedily, and of using local, if not general, bleeding in acute inflammatory disease. There can, I think, be little doubt as to the marked influence for good of calomel and opium in acute inflammation of serous membranes; and it would require very little persuasion to induce me to prefer this mode of treatment to that by iodide of potassium in acute spinal meningitis; and, further, I can readily believe that in such a case recovery

would be promoted by a judicious abstraction of blood. I have twice seen symptoms, so closely resembling those of acute spinal meningitis as not to be distinguished from them, disappear coincidently with the occurrence of local hæmorrhage, once from piles, once in the form of menstruation; and I can well believe that a similar result might be furthered by the application of leeches around the anus or to the cervix uteri—to these parts rather than to the back, because their vessels would seem to communicate more directly with the deep spinal vessels. It is very probable, however, that the time will soon pass in which depletion in any form, or depressing remedies of any kind, are required, and that the indications will rather be towards brandy, or ammonia, or turpentine, or ether, than towards the remedies which have been mentioned, for all acute diseases of the spinal cord would seem to have a rapidly devitalizing influence upon the system. In acute cases the catheter will be necessary to empty the bladder; in chronic cases, aching and stiffness of the limbs may point to friction and shampooing as likely means of relief. In every case there is sure to be some peculiarity to which attention must be directed if the plan of treatment be all that it ought to be; and, in short, every case must be treated on its own merits.

II. Myelitis.

Myelitis, or inflammation affecting the substance without involving the membranes of the cord, is a well-defined and not very uncommon disease. It may occur in an acute or in a chronic form: it may be general or partial: and, to say the least, its features are quite as well marked and distinctive as those of spinal meningitis.

1. Symptoms.—As an instance of acute myelitis, and as a text for what has to be said under this head, I take the notes of the case of a hospital patient under my care some time ago.

Case.—Charles K., a draper's assistant, twenty-six years of age, unmarried, a patient admitted into the National Hospital for the Paralysed and Epileptic on the 9th of January, 1864.

(*a*) The chief symptoms complained of are paralysis and anæsthesia below the waist, a disagreeable feeling of tightness around the waist, inability to pass water, involuntary stools, and pain in the left side of the chest. Above the waist, the power of movement and the power of sensation are natural; below the waist, all the voluntary muscles are entirely paralysed, and the sensibility to pain, to tickling, to differences of temperature, as well as to touch, are completely lost. Pressure along the spine is felt above the point to which the anæsthesia reaches, but not below it, and where felt the patient bears it without wincing. In other words, there is no tenderness on pressure in that part of the spine which preserves its sensibility. The feeling of warmth produced by passing a sponge soaked in moderately hot water along the spine is felt above the point to which the anæsthesia reaches, but not below it; and, where felt, the feeling of heat is natural, except at the line of junction between the sensitive and insensitive parts, and there the feeling produced is that of burning. Moreover, the warm sponge produces the same feeling of burning all around the body in the course of this line of junction, and thus it is plain that this local over-sensitiveness to heat is not confined to the spine. No reflex movements are produced by tickling the soles of the feet. The alæ nasi work very much, the lips are somewhat dusky, the lower intercostal muscles are motionless, and the accessory inspiratory muscles are in full work; the air-passages (especially on the left side) are loaded with phlegm, the pulse is hurried and weak, the skin is moist and somewhat cooler than natural, and the voice is so low as to be scarcely audible. A cough of the feeblest sort is almost incessant, but the expiratory power at command is altogether insufficient to bring about the expectoration which is so much needed. All appetite is gone, but food can be taken, and there is no thirst, or none to speak of. The urine, which is acid, and of the specific gravity of 1015, has to be drawn off by the catheter. There is no priapism. A stool has just passed without the patient being aware of it until his nose took account of the accident.

(*b*) A week ago, on awakening from a short nap, the patient found that his toes had gone to sleep, and that he had to "take long breaths." Instead of passing off, the feeling of tingling spread rapidly from the toes to the feet, from the feet to the legs, from the legs to the thighs, until it reached the seat, becoming less and less endurable as it spread, and being at last accompanied by a feeling of tightness around the waist and around the left instep, and by a state of restlessness which made it scarcely possible to sit still for a moment. After suffering in this way for a couple of hours, an attempt to pass water, which failed altogether, was followed by an almost intolerable uneasiness at the end of the penis, and by a sudden weakness in the legs which made it necessary to remain on the bed upon which he had fallen. Up to this time there had been no difficulty in standing, or walking, or even in going up and down stairs. A friend of the patient's now present says:—"I saw him on the evening of the day on which he was attacked, a couple of hours or so after he had been obliged to take to his bed. I thought he was suffering from severe rheumatic pains. For some hours those pains were excruciating. I had never before seen any one suffer so much. He tossed about in dreadful agony: he roared out with pain often, and, when not roaring, he groaned." Having thus passed seven or eight miserable hours, he fell asleep, and slept until breakfast-time next day. Upon waking in the morning he could neither move his legs nor empty his bladder; he had lost all feeling below the waist, and all the miserable feelings which had kept him in a state of continual unrest before he fell asleep were gone. On inquiring whether these feelings were of the character of pain, he says, "No, not exactly; worse than pain, one continued numb stinging feeling, as if the parts were asleep;" so that the friend's words which have just been given must be taken as meaning not exactly what they seem to mean in this particular. For the six days preceding his admission to the hospital a state of imperfect priapism was apt to come on of itself, or to be brought on by introducing a catheter to draw off the water, and this is the only point remaining to be noticed here, for in other respects the condition seems to have remained stationary, except, perhaps, that a little ground was lost every day.

The patient seems to have come of a healthy family, and, though never very strong, to have himself always enjoyed tolerably good health. He was confined to the house for a few days about two months ago by "influenza," and this is the only illness of any kind he remembers to have had. He says, "I was fatigued by a long walk on the day I was taken ill, and for a month and more I had felt more tired in my back and legs than usual in an evening, and more rheumatic—less up to the mark;" and also, "My back always ached at the end of the day's work, and so did my legs, and I was always glad to go to bed soon, for in bed I was comfortable:" and besides these statements there appears to be nothing at all calculated to throw light upon the history of his present malady.

Jan. 10.—Early this morning, after a sleepless night, a severe rigor commenced in the right arm, and then extended first to the back, and afterwards to the whole body. This rigor continued a full quarter of an hour, and was followed by profuse perspiration. During its continuance the paralysed parts were very cold: after it had ceased the warmth returned, and brought with it a considerable mitigation of the cough and trouble of breathing. Indeed, after the establishment of reaction, difficulty of breathing ceased to be an urgent symptom, except for a moment or two after waking from an occasional and very brief doze. The anæsthesia in the trunk has mounted full an inch higher since yesterday, but it has not extended to either of the upper extremities. Priapism occurs frequently. The pulse is 150; the respirations are 36 in the minute.

Jan. 11.—There has been no sleep in the night. The engorged condition of the lungs has gained headway, and the harassing suffocative cough has returned. Hiccough is frequent and distressing. Once during the day the passage of the catheter was obscurely felt, this being the first sign of feeling in this part since the commencement of the illness. The urine is decidedly acid. The electro-contractility and electro-sensibility of the paralysed muscles are annihilated.

Jan. 12.—For the last twenty-four hours the increased difficulty of breathing attending sleep has caused the patient to wake immediately if he for a moment forgot himself. "I can't breathe except I keep awake," he said in a voice scarcely audible; and also, "I hope I have not long to live." The passage of the catheter is still obscurely felt, and the escape of flatus and fæces is perhaps not so entirely unfelt as it has been since the commencement of the illness. In other parts the anæsthesia, like the paralysis, remains as complete as ever. The urine is still acid, distinctly so. For the last twenty-four hours there has been no priapism, and scarcely any cough. At present hiccough is almost constant, the pulse is fluttering, the hands are cold and clammy, and, in short, the signs of the near approach of death are not to be mistaken.

Jan. 13.—The patient lingered through the night, and died about daybreak; his mind unhappily remaining too clear to the very last.

The notes of the post-mortem examination are as follow:—

Jan. 14, 4.30 P.M.—Rigor mortis is fully established everywhere. The dependent parts present considerable signs of suggilation, especially along the course of the spine, and there is incipient breaking of the skin on both the nates. The arachnoid covering of the cord everywhere is clear, smooth, and without any traces of inflammation. The outside of the lumbar enlargement is curiously nodulated. On making a longitudinal section, the whole substance of the cord, from the brachial enlargement to its inferior extremity, is found to be of a yellowish red colour, softened in a remarkable manner, and in the lumbar region almost like cream in consistence. Several small patches of extravasated blood are scattered in the softened structure, these patches being undefined in outline, more numerous in the lumbar than in the dorsal region of the cord, and situated chiefly in the posterior columns. The red discoloration which has been mentioned is most marked in the neighbourhood of these patches. The examination did not extend further, the friends of the patient consenting to it only on condition that it should be thus partial.

Jan. 15.—On examining some portions of the diseased cord under the microscope, the natural structure is found to be altogether broken down, and mixed up with blood-corpuscles, exudation granules, and (in fewer numbers) pus-corpuscles.

With a view to arrive at a knowledge of the general features of myelitis, I select as the principal points for comment in this particular case the following:—Paraplegic anæsthesia, ushered in by tingling or some similar sensation in the parts which eventually became anæsthetic; paraplegia ushered in by uncontrollable restlessness; a disagreeable feeling of tightness around the waist and elsewhere; absence of pain in the spine or extremities—of pain produced by movement especially; absence of trismus and other spasmodic or convulsive symptoms; retention of urine; involuntary stools; absence of pain on pressure (spinal tenderness) in any part of the spine; increased sensibility to differences of temperature, by which moderately warm or iced water gave rise to a feeling of burning instead of the natural feeling over the vertebra which marks the upper limit of the myelitis; annihilation of reflex excitability in the paraplegic parts; priapism; acidity of urine; comparative voicelessness; impeded respiration; engorgement of lungs and other viscera; tendency to bed-sores; loss of electro-contractility and electro-sensibility in the paralysed muscles; absence of head-symptoms; absence of fever.

Paraplegic anæsthesia, ushered in by tingling or some similar sensation in the parts which eventually became anæsthetic.—In this case the anæsthesia was developed suddenly during the first night's sleep; it was deep-seated as well as superficial; it implicated the sensibility to pain, tickling, and differences of temperature, as well as that of touch; it had a paraplegic distribution: and this would seem to be the rule in cases of acute myelitis. In chronic cases it is developed more gradually, and it may not extend to all the various forms of sensibility; moreover, it may in some instances be quasi-hemiplegic instead of paraplegic; but the rule in acute cases appears to be what it is found to be in this. The anæsthesia seems to be usually ushered in by tingling or by some analogous sensation, disagreeable enough, but not amounting to actual pain. In this particular case the preliminary sensation was not pain, but an unbearable "numb stinging," as if the parts were asleep, with a feeling of tightness around the waist, and around one of the insteps. In acute cases it is right to speak of anæsthesia as ushered in by tingling or some similar sensation, but scarcely so in chronic cases. In chronic cases, indeed, these anomalous sensations may never exactly come to an end, because in these cases the destruction of sensibility may never get beyond numbness—may never reach nearer to anæsthesia; that is to say, than dysæsthesia.

Paraplegia ushered in by uncontrollable restlessness.—The paralysis was thus ushered in in the case under consideration, and in six similar cases which have come specially under my own notice, by restlessness, and *not* by any more marked tremulous, convulsive, or spasmodic symptom. Neither does it appear that a different rule obtains in other cases, acute, sub-acute, or chronic. In the great majority of cases, no doubt, the paralysis has a paraplegic form, but in a few cases it is not so. In the great majority of cases, the paralysis is accompanied by numbness, but not absolutely in all. Sometimes, for example, as in the case in which the paralysing lesion is limited to a portion of one lateral half of the spinal cord,—the case about which enough was said in the preliminary remarks,—there is paralysis without numbness on one side, and numbness without paralysis on the other side. Several cases of this kind are on record, and the number of them which I have myself met with is sufficient to convince me that they are scarcely to be looked upon as out of order and exceptional. Sometimes, also, as in the case where the paralysing lesion is confined to a portion of one of the anterior columns, the paralysis may be divorced from numbness, and not only so, but it may be hemiplegic in its distribution; and in such a case it may, in fact, be no easy matter to say whether it is dependent upon a cerebral or upon a spinal cause. In some cases, also, the paralysing lesion may be so localized as to affect only, or chiefly, an arm on one side and a leg on the other side. Usually, however, the paralysis is distinguished by being associated with numbness, and by being paraplegic in its distribution.

A disagreeable feeling of tightness around the waist and elsewhere.—A feeling of circular constriction around the trunk, or around some part of an extremity, around the trunk especially, is so common as to deserve to be considered as an almost constant symptom in myelitis. I do not recall a case, acute or chronic, in which it was entirely absent at all times.

Absence of pain in the spine and extremities—of pain produced by movement more especially.—In chronic cases of myelitis, Dr. Brown-Séquard speaks of "a constant pain in the part of the spine corresponding to the upper limit of the inflammation of the cord" as a characteristic symptom; but I question very much whether this statement is in accordance with well-sifted clinical facts. Pain, either in the spine or elsewhere, is not mentioned, for example, in the nineteen cases, acute or chronic, given by Ollivier, except in three; and of these three the myelitis was complicated with meningitis in two, and in the one remaining the symptoms justify the presumption (and there was no post-mortem examination to set it aside) that the same complication existed. At any rate, it is certain that there is not in uncomplicated myelitis that severe pain in the back and limbs which is brought on or aggravated by movement in spinal meningitis.

Absence of spasmodic symptoms.—Ollivier speaks of continuous contraction of the limbs as being met with "assez ordinairement," in chronic myelitis; but the cases cited by this excellent observer do not substantiate this statement. Thus, out of nineteen cases of myelitis, complicated and uncomplicated, acute and chronic, there are three only in which these contractions were present, and not one of the three can be cited correctly as a case of myelitis. In one of the three (No. 87) the sensibility was intact, and the disease of the cord confined almost exclusively to the anterior column; in another (No. 93) there was obtuse sensibility, and the disease was chiefly in the grey matter; and in the third (No. 94) sensibility remained, and there was no post-mortem examination to show what the disease in the cord really was. In each one of these cases, also, there were "head-symptoms" which do not figure in uncomplicated myelitis. Again, prolonged contraction of the extremities is a not unfrequent symptom in cases in which there is neither myelitis nor spinal meningitis—cases which properly come under the head of "spinal irritation," and about which more will have to be said in another section of this article. In these cases the contraction, instead of pointing to inflammation of the cord or its membranes, is really no more than one of a series of so-called hysterical phenomena. It is a sign of functional disorder only, and that it is so is evident (these among other proofs) in the sudden and complete way in which it passes off, as well as in the fact that it does not leave behind it any permanent organic traces. It depends, as it would seem, upon a state of irritation in some part of that track in which irritation gives rise to prolonged spasm—a state issuing, it may be, now and then in inflammation, but in itself, so far as the condition of the blood-vessels is concerned, diametrically opposed to inflammation. Nay, even in those exceptional cases of myelitis in which there is increased reflex excitability in the paralysed limbs, it is difficult to connect these spasmodic symptoms with the inflammation. Dr. Brown-Séquard says: "When the dorso-lumbar enlargement is inflamed, reflex movements can hardly be excited in the lower limbs, and frequently it is impossible to excite any. On the contrary, energetic reflex movement can always be excited when the disease is in the middle of the dorsal region, or higher up." And again, when speaking of the reflex convulsions which may happen in the cases where the inflammation is in the middle of the dorsal region or higher up, he says, "Convulsions do not take place at the beginning of the inflammation, but some time after, and they recur by fits for months and years after." And this is precisely what happens. In a word, the truth would seem to be that these reflex spasmodic movements must be referred, *not* to inflammation in the lumbar enlargement of the cord, nor yet to inflammation higher up in the cord; for in this case, to repeat what has just been said, "the convulsions do not take place at the beginning of the inflammation, but some time *after*, and they recur by fits for months and years *after*." They happen, as it would seem, *after* the inflammatory disorganization has interrupted the continuity of the cord, and produced a state of things analogous to that of a guinea-pig, or other animal, whose spinal cord has been cut across experimentally—a state of things of which increased reflex excitability in the paralysed parts is one of the consequences. Nor is a different conclusion to be drawn from the occasional presence in the paralysed muscles of a state which is analogous to or identical with the "late rigidity" of Todd. This "late rigidity" is very different from "early rigidity." In "early rigidity" the electro-motility of the muscles is increased, and the muscles relax during sleep, and to a less degree under the influence of warmth. The muscular contraction is evidently of the nature of spasm. In "late rigidity," on the contrary, the muscles are wasted, their electro-motility annihilated, and sleep or warmth do not tell in causing relaxation. This form of muscular contraction, indeed, if not identical with rigor mortis, is, as it would seem, more akin to this state than to spasm. In the case of myelitis which serves as my text, there was none of the painful muscular rigidity produced by movement which is so prominent a symptom in spinal meningitis. There was, indeed, no spasmodic symptom of any kind, with the exception of the rigor which ushered in the extension of the disease on the day after the admission of the patient to the hospital. And this absence of spasmodic symptoms would seem to be the rule in all cases of myelitis, acute or chronic. In children, it is true, myelitis may be ushered in by convulsion—in which case the convulsion manifestly represents the rigor which may usher in myelitis in adults, and as manifestly belongs to the precursory stage of irritation, and not to the state of actual inflammation—but even in children, unless there be some meningeal complication along with the myelitis, this preliminary convulsion would seem to be a rare phenomenon.

Want of control over the bladder.—This appears to be the earliest as well as the most constant of the symptoms of myelitis. It usually depends upon paralysis of the accelerator urinæ and compressor urethræ, but now and then it would seem to be connected, for a while at least, with a state of spasm in the latter of these muscles, in which case the dribbling away of the water or the introduction of a catheter will sometimes produce marked reflex spasms in the legs. I remember one case—a case in which the myelitis seemed to have interrupted the continuity of the cord high up in the back—where an attempt to use the catheter often gave rise to strong reflex spasms in both legs, and to a state of spasm in the urethra strong enough to prevent the passage of the instrument.

Want of control over the rectum.—In myelitis, paralysis of the sphincter ani is usually associated with paralysis of the accelerator urinæ and compressor urethræ. Now and then also, the sphincter ani, instead of being paralysed, may be in a state of reflex spasm: thus, in the case to which I have just referred, the administration of an enema was sometimes rendered impossible by the spasm set up in the sphincter ani and in the femoral muscles by the pipe.

Absence of local spinal tenderness.—As in spinal meningitis, so in myelitis, absence of tenderness on pressure in any part of the spine would seem to be the rule, and not the exception. Ollivier, speaking of pain in the back in myelitis, says, "Elle n'est jamais rendue plus aiguë par la pression," and my own experience in the matter is, without question, to the same effect.

Altered sensibility to heat and cold, by which a feeling of burning is felt when a sponge soaked in moderately warm water or a piece of ice is applied to the spine immediately above the seat of inflammation.—Several years ago it was pointed out by Mr. Copeland that, when a sponge soaked in water a little above the temperature of the blood was passed along the spine from above downwards, it gave rise to the natural feeling of heat until it reached the inflamed part, and that then this feeling changed to that of burning: and more recently Dr. Brown-Séquard has shown that a similar result is arrived at by passing a piece of ice down the spine, the natural feeling of cold being felt until the inflamed part is reached, and then an unnatural feeling of burning. In many cases, no doubt, all this would seem to be quite true, but not in all, perhaps not in the majority; and therefore it is impossible to look upon the feeling of burning thus produced as more than an occasional occurrence in myelitis.

Annihilation of reflex excitability.—What has to be said under this head has been anticipated when speaking of the absence of spasmodic symptoms in myelitis. It has indeed been seen to be the rule for all reflex movements to be annihilated or greatly weakened in the paralysed parts, and that the apparent exceptions to this rule are to be explained, not by referring the increased reflex movement to myelitis, but by supposing the inflammatory disorganization to have interrupted the continuity of the cord and produced a state of things analogous to that of a guinea-pig whose cord has been cut across for experimental purposes.

Diminution of electro-motility and electro-sensibility in the paralysed muscles.—Except in those few, very few, cases in which the reflex excitability is increased, the electro-motility and electro-sensibility of the paralysed muscles are invariably diminished in myelitis. Where the reflex excitability is increased the electro-motility may also be increased, and so also may the electro-sensibility, but more generally the increase in the former property is without a corresponding increase in the latter. The paralysed muscles are wasted in almost all cases, and relaxed also, except in those few cases in which the paralysis has lasted for a very long time and become associated with that state of "late rigidity" which, sooner or later, is often found to seize upon paralysed muscles. Marshall Hall noticed the impairment of irritability in spinal paralysis, and was of opinion that an opposite state of things existed in cerebral paralysis. As was pointed out by Todd, however, this supposed distinction between spinal and cerebral paralysis does not hold good, the simple fact being that in the great majority of cases of cerebral paralysis the irritability of the paralysed muscles, instead of being increased, is either not materially altered or else more or less diminished—most generally diminished in a very marked degree. In a word, the investigations of this very accomplished physician show most clearly that in cerebral paralysis the irritability of the paralysed muscles is only increased in those comparatively few cases in which the paralysis is associated with "early rigidity."

Priapism.—It is difficult to attach any diagnostic value to this symptom. As in acute spinal meningitis, so in acute myelitis, it is sometimes present and sometimes absent, less frequently present in the latter affection perhaps than in the former.

Acidity of the urine.—Dr. Brown-Séquard says: "One of the most decisive symptoms in myelitis is alkalinity of the urine. There is no patient attacked with myelitis in the dorsal region of the cord whose urine is not unfrequently alkaline. At times, especially after certain kinds of food, the urine is acid, but the alkalinity soon returns." And no doubt the urine is very generally alkaline in myelitis, especially in those cases in which the paralysis of the bladder has led to secondary disease of this organ; at the same time, as in the case under consideration, the urine is too often acid to make it possible to insist upon alkalinity of the urine as a necessary feature in myelitis.

Dyspnœa.—Difficulty of breathing was a very urgent symptom in the case which serves as my text, and so it must be in every case where respiratory muscles are so gravely implicated in the paralysis, and where the lungs are so much engorged. Indeed, the usual way in which myelitis proves fatal is by compromising the sufficiency of the respiration. Now and then, especially when chronic inflammation affects the higher regions of the cord, the difficulty of breathing may occur in paroxysms not unlike those of asthma, but usually the difficulty shows itself rather as simple shortness of breath,—shows itself in a way which supplies another proof of the absence of the spasmodic element in the history of myelitis.

Want of power in the circulation.—There is little or no sympathetic fever in the most acute form of myelitis; and in the ordinary chronic forms, the feeble pulse, the œdematous condition of the paralysed extremities, the disposition to passive engorgement in the lungs and elsewhere, and other symptoms of like meaning, show very plainly that the state of the circulation is eminently asthenic. It would even seem as if there were something in the very fact of myelitis which has a positive influence in subtracting power from the circulation—which exercises a devitalizing influence upon the system generally.

A tendency to bed-sores, wasting, and other signs of defective nutrition in the paralysed parts.—Sooner or later, generally at a very early date, a marked disposition to bed-sores in places where paralysed parts are subjected to pressure is apt to show itself in myelitis, and so also are other signs of defective nutrition in the same parts, such as œdema, dryness and scurfiness of the skin, and a wasted and flabby state of the muscles. So marked, indeed, is this impairment of nutritive power in these paralysed parts, that it is only by very great care that bed-sores and the other lesions which have been mentioned can be prevented.

Absence of head-symptoms.—In cases where acute myelitis attacks the higher portions of the cord, there may be, and there in all probability will be, various " head-symptoms "—vertigo, singing in the ears, grinding of the teeth, delirium, convulsion, coma, or others—but these cases, to say the least, are not common. Whether acute or chronic, indeed, myelitis is much more apt to attack the lower portions of the cord than the upper, in this respect differing from spinal meningitis; and when it attacks the upper portions of the cord, and its symptoms present cerebral complications, the chances are that the case is not simple myelitis, but myelitis with more or less spinal meningitis in addition.

When the cord is affected generally, the symptoms of myelitis will not differ greatly from those which are present in the case which has been given; when the inflammation is more localized, the symptoms will vary accordingly. If, for example, the inflammation be limited, as it usually is, to the lumbar enlargement of the cord, the level of the paralysis and anæsthesia will be proportionally low down; and if the extreme end of the cord only be affected, it is possible that the legs may escape altogether, and the bladder and anus be alone at fault. As indeed the level of the inflammation in the cord falls or rises, so must the level of the paralysis and anæsthesia fall or rise also.

Exaggerated reflex movements in the inferior extremities will also (in all probability) be associated with the paralysis and anæsthesia, if the lower part of the cord be sound and the inflammation confined to a portion of the cord higher up. Again, the symptoms which are present when the inflammation is limited to a part only of the thickness of the cord will be different in many respects from those which are met with when the whole thickness is affected. If, for example, a portion (the upper half-inch of their course excepted) of the anterior columns be affected solely, there would be paralysis without anæsthesia; or if the posterior columns were alone affected, there might be incoordination of movement and some hyperæsthesia instead of paralysis and anæsthesia. In short, the variations of symptoms, which occur where myelitis is restricted to particular parts of the cord, can only be properly intelligible to him who has clear notions respecting those physiological matters which were glanced at in the preliminary remarks,—which were then glanced at chiefly in order to avoid perplexing physiological digression and discussion in the present place among others. I will, therefore, assume that what was said in the preliminary glance at some points in the physiology of the spinal cord, will serve to explain sufficiently the variations of symptoms which may be expected to exist when the integrity of particular parts of the spinal cord is destroyed by myelitis or in any other way: and, for the rest, I will only say that myelitis may be chronic and subacute as well as acute in its course, and that these several varieties interblend insensibly the one with the other.

2. Post-mortem Appearances.—Myelitis may result either in softening or in hardening of the spinal cord. Most frequently the cord is broken down, reduced to a yellowish or reddish cream-like consistence; the colour, derived from the admixture of pus or blood-corpuscles, being more yellow or more red according as the one or the other of these corpuscles predominates. This softening may affect the whole thickness of the cord, or certain parts more than others, the grey matter especially; it may extend from one end of the cord to the other, or it may be confined to certain regions, in which latter case the part most likely to be affected is the lumbar enlargement; and it would often seem to have its starting-point in the central grey matter, which is the most vascular part of the cord. In the first stage of myelitis this central grey matter has a rosy or vinous tinge, which is not natural to it; it is plainly more vascular than it ought to be; and, in short, it has undergone the very same change which is met with in the grey matter of the brain in encephalitis. Sometimes the spinal cord is considerably swollen, and sometimes the surface may have a nodulated appearance in certain parts, from the membranes having yielded at these points to the blood which may have escaped, or to the pus or other fluid which may have collected, underneath. Not unfrequently small collections of blood are met with in the softened nerve tissue, espe-

cially in the position of the central vessel, so that the first impression upon opening the cord may be that of hæmorrhage rather than that of myelitis. One remarkable feature of inflammatory softening, says Dr. Todd, is that "it exhales a marked odour of sulphuretted hydrogen, and so indicates a rapid advance of putrefaction;" and again, "It is a fact deserving of attention that the substance of the spinal cord softens very rapidly after death, the lapse of half an hour, during which the nervous substance has been exposed to the air, often producing a manifest alteration." Indeed, there are reasons for believing that the amount of disorganization met with in the cord after death does not necessarily represent the exact amount which existed during life, and that a cord which is found to be broken up after death almost utterly, may have retained during life sufficient integrity to allow of the transmission of certain sensitive and motor impressions. On this view the return of slight sensation in the urethra and rectum shortly before death, and the preservation of the power of moving and feeling in the arms, which were noticed in the case which serves as my text, are not altogether unintelligible.

Induration, the other result of myelitis, is looked upon by some as a stage always preceding softening, but it would rather seem to mark, as Ollivier supposed, a less acute form of inflammation. In it the fibrinous products of the inflammation seem to have been more organizable. The cord thus indurated varies greatly in appearance; it may be almost as pale, bloodless, crisp, and hard as cartilage; it may be more or less red and vascular; and in either case, when examined under the microscope, its proper tissues are found to be broken up and destroyed almost as effectually as they are when the cord is softened. A cord which is indurated has usually a shrunken appearance, but it may be swollen considerably. There is no doubt an induration of the cord, as well as a softening, which cannot be referred to myelitis, and which must not be confounded with that which is the result of inflammation; but I must not stay to point out the differences, nor yet to do more than say that in myelitis there will in all probability be found, in addition to the signs which have been indicated, engorgement of the lungs, kidneys, and other viscera, possibly more marked vascular changes, with bed-sores, œdema, dry and scurfy skin, wasted muscles, and other signs of defective nutrition in the paralysed parts.

3. Causes.—Nothing very much to the point can be said under this head, and the only remark I feel called upon to make is this, that as in spinal meningitis a rheumatic habit has been found to figure more or less conspicuously among the causes of the malady, so here a like position would seem to be due to a strumous habit. I would also confess to a growing impression that myelitis may not unfrequently be connected more with excess of sexual indulgence than with any other single cause, but I cannot say that this impression has yet taken the form of a definite conviction.

4. DIAGNOSIS.—In dealing with the symptoms of myelitis it has been shown that these are very different from those of spinal meningitis—so different as to make it difficult to confound them, if only moderate care be taken in realizing them. In spinal meningitis the most prominent symptom is pain in the back and extremities, produced or aggravated by movement; in myelitis pain of any kind has scarcely a title to be reckoned among the symptoms, pain produced by movement certainly not. In spinal meningitis the sensibility is somewhat exalted, in myelitis it is abolished. In spinal meningitis there is muscular weakness, and the muscular movements are fettered by pain, but there is no true paralysis; in myelitis, paralysis is the symptom of symptoms. In spinal meningitis there is a state simulating trismus and tetanus, a state of muscular rigidity half voluntary as to its character, of which the object is to prevent certain movements which give rise to pain; in myelitis the muscles are limber, and there is usually an utter absence of any symptom akin to tremor, convulsion or spasm.

Nor need the symptoms of common paraplegia (resulting from chronic myelitis) be confounded with those of locomotor ataxy. In common paraplegia there is paralysis more or less marked of the lower extremities, and the nutrition and irritability of the paralysed muscles are, as a rule, unmistakeably impaired; not so in locomotor ataxy. In common paraplegia the paralysis extends to the bladder and sphincter ani, and the sexual power is greatly weakened, if not altogether abolished; not so, or not to anything like the same degree, in locomotor ataxy. In common paraplegia the characteristic neuralgic pains of locomotor ataxy are wanting, and numbness is nothing like so prominent a symptom as in the ataxic disorder. In common paraplegia, where walking is possible, the gait—instead of being precipitate and staggering, the legs starting hither and thither in a very disorderly manner, and the heels coming down with a stamp at each step, as in locomotor ataxy—is hampered and slow, each leg being brought forward with evident difficulty, even with the help of an upward hitch of the body on the same side, and the part of the foot first coming in contact with the ground being, as a rule, not the heel, as in ataxy, but the toes. In common paraplegia, impairment of sight or hearing, or strabismus, or ptosis, or injection of the conjunctivæ, or contraction of the pupils, frequent if not constant symptoms in locomotor ataxy, form no part of the history. In fact, in these respects, and in others of minor importance which might be mentioned, the histories of common paraplegia and locomotor ataxy are so different that it is not easy to see how, with only a moderate amount of care, the two can be confounded.

Now and then, it is true, instances occur in which it is not so easy to distinguish this gait of common paraplegia from that of locomotor ataxy—cases in which the weakened muscles contract somewhat spasmodically when put in action, but, as a rule, the gait in common paraplegia and in locomotor ataxy is sufficiently characteristic to make it difficult to confound these two affections.

In cases where the myelitis is confined to the posterior columns of the cord, the symptoms will be those of locomotor ataxy rather than those which have been ascribed to myelitis; for, so far as the production of symptoms is concerned, it is of no moment whether the disease disorganizing the posterior columns be inflammatory or non-inflammatory, acute or chronic: and in other cases of local myelitis symptoms are sure to be present which cannot fail to lead to a correct diagnosis, if what was said in the preliminary remarks upon the physiology of different parts of the spinal cord be borne in mind in interpreting them. Indeed, with what is now known of the physiology of the spinal cord, there need not be much difficulty in determining the whereabouts of local mischief in the cord.

That myelitis cannot well be confounded with other spinal disorders—spinal congestion, tetanus, spinal irritation, and the rest—will be seen readily enough when a clear idea of these disorders has been realized, and only then; and this being the case, it is best to waive these questions in diagnosis until the fitting opportunities for dealing with them present themselves.

5. Prognosis.—Acute myelitis affecting any considerable extent of the spinal cord is, without doubt, a very grave disorder. It may be fatal in fifteen or twenty hours, and it is seldom that life is prolonged beyond the end of the second week. Instances of recovery are on record, it is true, but these are very few in number, and of them there is, perhaps, no single one in which the correctness of the diagnosis may not be impugned. Even chronic myelitis is a very grave disease; for though life may be prolonged, especially where the disease is confined to the lower part of the cord, the mischief once done seems to be in a great measure irreparable. At the same time it is only right to say that of late years the results of treatment have been much more satisfactory, and that it is possible now to hope where there was little room for hoping formerly.

6. Treatment.—There appears to be little room for what is called active treatment even in acute myelitis. The inflammation is evidently of a very low type, and, reasoning from what is known of its beneficial action in erysipelas and in some other low forms of inflammation, it seems to me that sesquichloride of iron would be likely to be of more real service than iodide of potassium. Indeed, I should be disposed, until I know of a better plan, to trust chiefly to full doses of this preparation of iron, to food and wine, and to the position recommended by Dr. Brown-Séquard for draining away blood from the spine—a position in which the patient is made to lie upon his abdomen or side, with his hands and feet in a somewhat dependent position.

With respect to the good or bad effects of belladonna, or ergot, of strychnia, it is not very easy to arrive at a satisfactory conclusion. I agree with Dr. Brown-Séquard in thinking that belladonna and ergot may have the effect of counteracting a hyperæmic condition by causing contraction in the vessels, and that the vessels of the spinal cord may, perhaps, respond most readily to their action, but not as to the indications for employing these remedies. Pain and spasm are, to Dr. Brown-Séquard, signs of hyperæmia: to me, except the pain produced by movement, they are signs of irritation only—of a state which is connected, not with hyperæmia, but with anæmia, a state of contraction of the vessels which may pass into relaxation, but which need not necessarily do so; and, therefore, to me pain and spasm, instead of being indications for the employment of belladonna or ergot, are in very deed contra-indications. Nor can I agree in thinking that strychnia acts by increasing the amount of blood in the spinal cord and in its membranes, and that on this account it is contra-indicated in hyperæmic conditions of these parts. Strychnia, without doubt, produces tetanic spasms and other unequivocal signs of spinal irritation, but it is begging the question altogether to suppose that the strychnia increases the amount of blood in the cord and its membranes, that this increase of blood augments the vital activity of the cord, and that the spasms and other signs of irritation attest this augmentation of vital activity. Indeed, so far from this being a necessary conclusion, all the evidence presented in the preliminary remarks, as it seems to me, points in the opposite direction, and connects the state of irritation of which the spasms are the signs, not with a hyperæmic condition, but with an anæmic; and most assuredly I know of nothing in the history of myelitis or spinal meningitis which is calculated to invalidate this conclusion. Moreover, the investigations of Dr. Harley upon the action of strychnia upon the blood go to show that this action is really equivalent to loss of blood in that it directly interferes with the proper arterialization of the blood. In a word, I cannot find any fundamental difference between the action of belladonna, ergot, and strychnia upon the blood-vessels, neither can I understand why strychnia, properly used, might not be of as much service as belladonna or ergot in lessening hyperæmic condition of the cord. For my own part, however, I onfess to a feeling which makes me hesitate to employ either elladonna, or ergot, or strychnia in myelitis, or in any analogous ondition, until I know more of their action, or until I have more unequivocal empirical evidence of the good resulting from heir use.

In chronic cases the one grand indication of treatment, as it seems me, is to improve the nutrition of the cord, and the medicines best lculated to carry out this indication are cod-liver oil, sesquichloride of on, phosphorus in one form or other, arsenic, and possibly bichloride mercury, which latter preparation, when properly used, I believe to tonic and antiseptic in a high degree, and in many respects much more analogous in its action to arsenic than to any of the proto-compounds of mercury in common use.

The local means for promoting the recovery of the paralysed muscles are certainly of not less importance than the general means, possibly of much greater importance, and these local means are very various. The efficacy of frictions and shampooings appears to be indisputable. The efficacy of proper movements can only be doubted by those who are unacquainted with the results arrived at by the "movement cure," and by systematic movements of one kind or another, with or without the help of mechanical apparatus. The efficacy of faradization has been abundantly proved, and there is good reason to believe that this is not the only mode of using electricity which will be of great service; that in fact statical positive electricity, or the interrupted galvanic current, or the application of the galvanic current in such a way that the paralysed nerve is acted upon chiefly by the positive pole—a mode of using electricity about which I have spoken elsewhere, and which I have used extensively during the last five or six years—will often be of great service in proper cases. Indeed I should think that the treatment was wanting in very essential particulars if these local means, one and all, were not associated with the general means of treatment, and employed systematically and perseveringly; and especially I should regard it as a great blunder if these local means were deferred so as to allow the paralysed muscles to lose what when lost is not easily recovered,—that is, their irritability and healthy organization.

There are also other local measures which are of great service in the treatment of paralysis, and one of these to which I am disposed to attach especial importance is to protect the paralysed parts from cold. In many cases, as is well known, these paralysed parts are cool, and in not a few instances, where the paralysis is incomplete or associated with early rigidity, this paralysis and rigidity is greatest when these parts are coldest. For example, it is no uncommon thing for a partially hemiplegic patient whose paralysed fingers are contracted, stiff, and altogether useless when acted upon by cold, to be able to open his hand and use his fingers with comparative freedom when the hand is warm in bed, or placed in a warm bath, or held a while before the fire. At any rate, I have long been satisfied that the well wrapping up of the paralysed parts in woollen, or silken, or india-rubber coverings is an important help in treatment.

It would also seem that good of the same kind, much good, may be got from an exhausting apparatus made on the principle of Junot's boot. The effect of such an apparatus, properly used, is to make the paralysed parts warmer at the time, and to enable them to preserve this warmth for a considerable time,—to produce a change in the circulation, which must have a good effect upon the nutrition and irritability of the paralysed muscles.

It is also more than probable that electricity may be of service in improving the condition of the circulation in the paralysed parts, for an increased feeling of warmth in the paralysed parts is the result of faradizing these parts, or of electrifying them with statical electricity; indeed I have been more than once disposed to think that the beneficial effects of electricity in the resuscitation of paralysed parts are as much brought about indirectly by changes produced in the circulation as by changes wrought directly in the nerves and muscles.

As regards the necessity for tenotomy and the use of orthopædic apparatus in certain cases, it is difficult to speak to any good purpose. I shall have to refer to these subjects when speaking of infantile paralysis, and here I will only say, that in many cases, in children especially, the cure will be greatly facilitated by tenotomy and orthopædic apparatus, and that it is not always easy to decide between the cases in which these measures are desirable and those in which they are not desirable.

III. Spinal Congestion.

Spinal congestion, or plethora spinalis, is not less definite in its history than myelitis or spinal meningitis, neither is it of less practical interest. In the sequel, indeed, it may appear, not only that spinal congestion is fully entitled to the place which has been assigned to it in the catalogue of diseases, but also that it really comprehends more than one spinal disorder which is now known under a different name.

1. Symptoms.—As an instance of well-marked spinal congestion, I take the notes of a case under my care not long ago.

Case.—Mary L., aged 28, but looking very much older, married but never pregnant, was admitted into the Westminster Hospital on the 12th of June, 1866.

(*a*) With the exception of being able to turn her head on the pillow and to move the fingers and toes a little, all power of voluntary movement appears to be wanting. The symptoms chiefly complained of are tingling in the tips of the fingers and toes, a dull burning aching along the back and in the limbs, and a feeling of being "tired to death." If altered in anywise, the sensibility to touch, pain, tickling, and differences of temperature, is somewhat more acute than natural. The spine is nowhere tender on pressure, but the dull burning aching in this region is increased by the application of a sponge soaked in hot water. The soles of the feet may be tickled without giving rise to undue reflex movements. The bladder and bowels act properly. The mind is not at all affected. The state generally is evidently one of great exhaustion and prostration without fever, the pulse being quick, unsteady, and very compressible, the respiration shallow, and curiously interrupted by sighs.

(*b*) Three weeks ago, menstruation, which had only just begun, was suddenly checked by an alarm of fire. This was shortly before bedtime. The next morning, after a very sleepless and miserable night, the state had become very much what it now is. Up to this time the patient had never been obliged to remain in bed a single day on account of illness. She had often been weak and ailing, and she had suffered a good deal at the menstrual periods from pain and weakness in the back and legs, and that is all. She also appears to have sprung from a tolerably healthy stock.

(*c*) Within the first fortnight after admission to the hospital, the tingling in the tips of the fingers and toes came to an end, and so did the aching in the back and limbs. A week later the arms as well as the hands could be moved a little. At the end of six weeks the legs remained almost as helpless as at first, but the arms and trunk had so far recovered power as to allow of a change from the lying to the sitting posture without any great difficulty. At the end of twelve weeks it was possible to get out of bed, and, with the help of a stick, to move to the table in the centre of the ward. On the 3d December, five months after admission, the patient left the hospital convalescent. All this while the appetite was tolerably good, and the bladder and bowels acted properly. Now and then, in the progress towards recovery, especially about the menstrual periods, there were short relapses in which the tingling in the tips of the fingers and toes, and the aching in the back and limbs, came back, and the paralytic weakness of the muscles was almost as great as at first,—in which the ground already gained seemed all but lost. Now and then, also, the nights were disturbed by a distressing state of shortness of breath, not amounting to asthma. Before the legs recovered power their muscles were somewhat wasted, but not considerably so; indeed, neither here nor elsewhere was the paralysis accompanied by any marked wasting of the muscles, or by any appreciable impairment of electro-sensibility or electro-contractility. Moreover, any movement, whether active or passive, had always the effect of relieving rather than of increasing the aching in the back and limbs, when this symptom was present. The treatment pursued was chiefly rest, good living, hypophosphite of soda, nux vomica now and then in small doses, cod-liver oil, and faradization.

Assuming, as I well may, this to be a case of well-marked spinal congestion, I take as points of comparison between it and other cases of the kind, general and partial, these:—suddenness of access; incomplete paralysis in a paraplegic form; no numbness; tingling in the tips of the fingers and toes; no exaggeration of reflex excitability in the paralysed limbs; no want of control over the bladder and bowel; no spinal tenderness; aching in the back increased by warmth; pains in the back and limbs not increased by movement; no marked impairment of the electro-contractility and electro-sensibility, and no material wasting, of the paralysed muscles; no feverishness; breathlessness; no bed-sores; proneness to relapses.

Suddenness of onset.—To be well, or comparatively well, on going to bed and to be paralysed in the morning, as in the case which I have given, is no uncommon thing in spinal congestion. It is indeed the rule rather than the exception for the illness to be spoken of as a "stroke" by the sufferer.

Incomplete paralysis in a paraplegic form.—Paralysis, often all but complete, but never quite so, and taking the paraplegic form, must be looked upon as the rule in spinal congestion. The paralysis is decidedly paraplegic in the end, and it may be so from the beginning, but not unfrequently one leg or one arm is affected before the other, and occasionally the leg and arm of the same side may for a short time be affected, as in hemiplegia, before the disease extends to the leg and arm of the other side. Not unfrequently there remains a difference in the degree of paralysis on the two sides, one leg or arm being more affected than its fellow. In cases where the congestion of the cord is general the arms as well as the legs are paralysed, the former perhaps as much as the latter; but in the common run of cases, where the congestion is confined chiefly to the lumbar region of the spine, the legs are exclusively or chiefly affected.

No anæsthesia.—Numbness is a symptom of myelitis, but not of spinal congestion. In the latter disorder, indeed, instead of numbness there is occasionally a state of things which may be spoken of as hyperæsthesia: thus, in a case very like the one I have given, which came under my notice in private practice about three years ago, the weight of a single bed-sheet was distressingly heavy to the patient, and long-continued aching of the paralysed arms and legs was produced by handling them ever so lightly.

Tingling in the tips of the fingers or toes of the paralysed limbs.—This symptom is almost always present at one time or other, coming and going and staying a longer or shorter time, often, as it would seem, very capriciously. One is glad to get rid of it, for while it remains it is difficult altogether to put aside the fear lest the state of the cord should pass out of simple spinal congestion into the graver disease of myelitis.

No exaggeration of reflex-excitability in the paralysed limbs.—Increased disposition to reflex movement is usually regarded as one of the symptoms in spinal congestion. It is supposed that the greater afflux of blood to the spinal cord must bring with it greater reflex excitability. I believe, however, that this supposition is not at all borne out by the facts. I believe, indeed, that the moderate reflex excitability in the case under consideration is not at all exceptional, and that it is the rule in all cases of spinal congestion for this manifestation of muscular contractility to be, if altered at all, diminished rather than increased.

No paralysis of the bladder or sphincter ani.—In myelitis, paralysis of the bladder or sphincter ani, more or less complete, is a prominent symptom: in spinal congestion, on the contrary, these symptoms are absent, except in those mixed cases where there is reason to believe that some degree of myelitis is also present. In the case which I have given there was not the least want of control over the bladder or bowel from the beginning to the end.

No tenderness on pressure along the spine.—Absence of spinal tenderness I believe to be the invariable rule, not only in spinal congestion, but also in myelitis and spinal meningitis. I believe, indeed, that spinal tenderness is a sign of the presence of that functional disorder of the cord which is usually called spinal irritation, and that it does not accompany the graver diseases of the cord which have been named when they are uncomplicated with spinal irritation. Upon this subject I shall have more to say presently.

Dull aching along the spine increased by warmth.—I have noticed this symptom in three cases of well-marked general spinal congestion which have come under my own observation, and in many cases of partial congestion; and I am disposed to think that this will prove to be one of the points of difference between spinal congestion and spinal irritation. I have also noticed the same symptom in myelitis and spinal meningitis, and therefore I cannot regard it as having any special connexion with spinal congestion. In fact, so far as my experience goes, I can say that this symptom is likely to be met with in congestive or inflammatory diseases of the cord, but not in spinal irritation simply; and that in this latter case, the local application of warmth to the spine is more likely to relieve pain than to cause it.

Pains in the back and limbs not increased by movement.—This symptom has some claim to be regarded as constant. The aching would seem to go and come with the congestion; and the fact, for fact it seems to be, that it is not increased by movement, may help to distinguish spinal congestion from spinal meningitis, for in the latter affection movement of the limbs, whether passive or active, is attended with pain in the parts moved and in the back.

No marked impairment of electro-contractility and electro-sensibility in, and no wasting of, the paralysed muscles.—In myelitis the paralysed muscles are prone to waste and to lose their electro-contractility and electro-sensibility, and herein, therefore, would seem to be a marked difference between this disorder and spinal congestion; for, so far as I know, the contrary state of things invariably holds good in spinal congestion.

No feverishness.—This is no special feature; indeed, fever would seem to have little to do with any affection of the cord, not even excepting meningitis in its most active form.

No bed-sores.—A marked disposition to bed-sores would seem to be the rule in myelitis, but not so in spinal congestion or spinal meningitis. Upon this point, more than upon many others, there is tolerable unanimity of opinion.

Shortness of breath.—Where the spinal congestion is at all general, this state of things may be readily accounted for by the paralytic weakness of muscles concerned in respiration. In the case which nerves as my text, the occasional shortness of breath is noticed as not amounting to asthma; and this is a point of some interest, for it may be supposed that the difficulty of breathing would have taken this form—would have had something of a decidedly spasmodic character—if the congested condition of the cord involved, as it is supposed to do, an exaggeration of reflex excitability.

Proneness to relapse.—Whether this may prove to be a constant feature in spinal congestion remains to be seen. That it is not an uncommon one is, to say the least, highly probable.

Spinal congestion varies greatly in its degree and in the extent of cord implicated. Limited to the lumbar region, and carried to a degree which produces, not paralysis, but weakness more or less approaching to paralysis in the legs, it is common enough ; indeed, many women seem to suffer from it before every menstrual period ; and between this partial and incomplete form and the general and complete form, of which the case which has been given is an instance, there are all possible grades of transition. It would seem to be most common in women, but it is not peculiar to the female sex or to any age. The onset of the disorder is generally sudden, in relapses as well as in original attacks ; and the cases do not at all divide themselves into acute and chronic as do the cases of many other disorders.

2. POST-MORTEM APPEARANCES.—These appearances are very vague and unsatisfactory, at most being simply some engorgement of the veins of the spinal cord and membranes, with some excess of the spinal fluid, both of which phenomena, as will be easily understood, are not very unlikely to escape detection unless the post-mortem examination be conducted with unusual care. With the exception of this engorgement and serous effusion, the only morbid sign which has been noticed (and this by no means constantly) is slight infiltration with blood of the cellular tissue exterior to the dura mater. In all uncomplicated cases, the structure of the cord and of its membranes is in nowise altered.

3. CAUSES.—As in the case which I have given, the suppression of the catamenia would seem to figure most conspicuously among the causes of spinal congestion, and next to this the cessation of hæmorrhage from piles. Beyond this it is difficult to single out any one cause which has a just claim to be considered as at all special : and, for the rest, nothing further need be said except this,—that spinal congestion is not unfrequently a consequence of pulmonary or abdominal congestion or inflammation—a consequence, perhaps, which has often more to do in compromising the safety of the patient than the primary disorder itself.

4. DIAGNOSIS.—Paraplegic paralysis is a symptom common to spinal congestion and myelitis, with this difference, that it is less complete in the former affection than in the latter. The paralysis is associated with anæsthesia in myelitis ; not so in spinal congestion. The control over the bladder and bowels is lost in myelitis ; not so in spinal congestion. The paralysed muscles are prone to waste and lose their

electro-contractility and electro-sensibility in myelitis; not so in spinal congestion. The absence of anæsthesia would seem, indeed, to connect spinal congestion more closely with spinal meningitis than with myelitis, and so also would the pain in the back and aching in the limbs; but the pain and aching in spinal congestion cannot well be confounded with the pain which is met with in spinal meningitis, for the pain in this latter affection is produced by movement and accompanied by rigidity, whereas the pain in the former affection is not produced and accompanied in this manner. Hysterical paralysis, so called, agrees with the paralysis depending upon spinal congestion in some respects, but not in others. It agrees in that the paralysed muscles are neither prone to waste nor to lose their electro-contractility; it disagrees in that numbness is a prominent symptom, more prominent even than the paralysis, and that the electro-sensibility of the paralysed muscles is either annihilated or very much diminished.

5. Prognosis.—Recovery is the rule, no doubt, in cases of spinal congestion, but there is no difficulty in finding cases in which the disease has been fatal, and quickly fatal too. In the partial form, affecting the lumbar portion of the cord only, spinal congestion may come and go quickly without any great damage being done; but in the cases in which the cord is more extensively and more profoundly affected, as in the case which has been cited, recovery may occupy a considerable time. Thus, of the cases recorded by Ollivier, No. 55 remained in hospital nearly five months, No. 56 two months, No. 57 three months, and No. 58 "assez longtemps." Recovery is slow, it may be, because time is required for the absorption of the excess of spinal fluid to which the state of spinal engorgement had given rise.

6. Treatment.—What has been said respecting myelitis must be supposed to apply here equally. Indeed, the only special remark which appears to be called for in this place is this,—that in cases where, as very generally happens, the spinal congestion can be referred to suppression of a menstrual or hæmorrhoidal discharge, the primary indication would appear to be the setting up of an equivalent discharge by applying leeches to the os uteri or to the anus.

IV. TETANUS.

Tetanus is unhappily no rare or unfamiliar malady. The name, from τείνω, I stretch, refers to that rigid and cramped condition of the muscles which is the most characteristic symptom, and which, in sober earnest, is suggestive of rigor mortis, not only *in posse* but actually *in esse;* for there are some cases in which, without any interval of relaxation, tetanic rigidity at once passes into cadaveric rigidity. Hydrophobia alone excepted, tetanus is at once the most appalling and the most perilous of all spasmodic diseases.

1. Symptoms.—As an instance of well-marked tetanus, I take the notes of a case which I happened to see from the beginning to the end ten years ago.

Case 1.—Patrick M——, a fair, slightly-built, delicate-looking man, unmarried, aged 27, the coachman of a gentleman then under my care. On the 21st of April, 1861, meeting him as I was leaving the house of his master, he took the opportunity of saying that he was not well enough to bring round the carriage, and of asking me what he had better do. What he complained of chiefly were a stiff neck and sore throat, with a feeling of weakness and illness. The stiff neck and sore throat made their appearance for the first time this day; the feeling of illness and weakness has been present for the last three days. The mouth cannot be opened so as to allow a fair look at the tongue, and a meal, it appears, has just been left unfinished, not for want of appetite, but simply on account of the difficulty experienced in masticating and swallowing the morsels. There is no feverishness.

P. M. ascribes his present indisposition to having been out with the carriage several hours in the wet and wind three nights ago, and he says further that he is liable to colds. Before speaking to me, he had taken some opening medicine which a chemist had prescribed and prepared for him, and he thinks that this dose may account for the fact of feeling so ill and weak at the present moment. Some simple treatment was recommended, and I took my leave, not at all divining what was so soon to follow.

April 22.—Receiving information that this poor fellow was very ill, I went round to see him at his lodgings. I found him strangely altered. His teeth were firmly and inseparably clenched, and he looked literally like an old man—so like, that his mother, who lived with him, said that she could have thought his father had come back to life if only his hair had been grey. His voice has also become so low and indistinct as to make it difficult to catch what he said. The medicine given by the chemist yesterday, it appears, has purged him violently several times in the night, and more than once while at stool he has been seized with acute pain in the pit of the stomach, which took away his breath, and made him think he was going to die. It was in the night, while at stool, that the jaws became closed. I wished him to go to the hospital, and he was willing to do so, but his mother would not consent. Eggs beaten up with brandy were ordered to be given repeatedly, and every three hours a draught containing five grains of quinine and half a drachm of Hoffmann's anodyne. I now noticed on one of the fingers, which was tied up in a piece of rag, a small wound, healing and apparently healthy, the result of a tear by a rusty nail about a fortnight ago.

On a second visit, later in the day, I found that repeated attempts had been made in the interval to give the food and medicine, but with very trifling success. There was no great difficulty in getting the food or medicine into the mouth, for almost all the teeth on the right side were gone, but the attempt to swallow brought on spasm in the throat, and on more than one occasion the spasm forced the greater part of what was taken back through the nostrils. And this difficulty was all the more distressing, because a feeling of hunger prompted the patient of his own accord to make frequent attempts to swallow. The chief complaint now was of a dragging pain at the pit of the stomach, piercing through to the back. In answer to a question whether he could sit up in bed, he said, "I think I am too stiff to do so," and then he tried to sit up, and succeeded after making two or three abortive attempts. While sitting up, I found that he could scarcely move his head, and that the muscles of the neck and back were very stiff and hard. I had only just noticed these phenomena when the noise caused by the upsetting of a chair brought on a fit of spasm, in which the patient was suddenly thrown backwards upon the bed with considerable force, and left resting upon his head and heels, in a state of complete opisthotonos—a state so complete as to make it possible for me to pass my hand under the loins without touching either the body or the bed. This severe spasm lasted not less than a couple of minutes, and the only muscles which did not seem to be implicated in it were the abdominal, those of the arms and hands, and those of the eyeball. In this spasm the complexion became dusky and livid, and the features altered in a frightful manner, the angles of the mouth being drawn upwards and outwards so as to give the expression known as the *risus sardonicus*, the set teeth being slightly uncovered, the nostrils spread, the eyes staring and prominent, the brow knit, the hair bristling,—the complexion and features became changed, that is to say, as they are changed in sudden suffocation. All this while, too, the skin generally was dusky and hot and drenched in perspiration. For some time after this spasm had passed off the patient remained moaning, and unable to speak audibly, and then he said, "that pain will kill me if it comes back." I noticed, also, that there remained after this spasm a state of tetaniform rigidity and contraction, by which no inconsiderable degree of opisthotonos was still kept up. The eggs and brandy and the medicine were ordered to be given by enema.

April 23.—Two attempts were made to administer the enemata ordered over night without success, the irritation of the pipe in each instance bringing on a fit of spasm; indeed, all that it has been possible to give since my last visit have been a few sips of wine and water. There has been no sleep whatever during the night. During the last eighteen hours several fits of spasm like the one described have occurred, and the permanent rigidity and contraction remaining between the fits have increased. The abdominal muscles, which were not at all implicated yesterday, are now as hard and stiff as those of the neck, back, and legs. The pulse is quick (about 140), weak, and somewhat irregular: the breathing is shallow, hurried, and frequently checked by gasps and catches, even when it is not interrupted by the fits of spasm.

No material change has taken place since the morning. On one occasion in the course of the day an egg beaten up with some brandy has been swallowed, but all other attempts to administer food or medicine, whether by the mouth or by the rectum, have been rendered abortive by the fits of spasm to which they gave rise.

April 24.—Again the night has passed without sleep, and to-day the constant tetaniform contraction has become almost universal. In fact, the only muscles which are not obviously affected are those of the hands, and tongue, and eyeball. The fits of spasm, also, are now more frequent and severe, being not more than fifteen or twenty minutes apart, and lasting until death from suffocation seems even more than imminent; they are brought on by the most trivial causes —an attempt to swallow, a draught of air, the simple straightening of the bedclothes—or they come on without any apparent cause. There is no improvement in the breathing and pulse, but if anything a change for the worse. During the fits the skin is hot, dusky, and drenched in perspiration: in the intervals it has an ominous coolness and clamminess. The mouth is full of viscid frothy saliva, and there is much thirst. While I was present a small quantity of dark urine was passed slowly and with some difficulty, and this appears to be the only time the bladder has acted for at least twenty-four hours. The pupils are large, especially in the paroxysms.

Shortly before I went again at the end of the day, there had been a momentary snatch of sleep, which had been abruptly brought to an end by an attack of opisthotonos, in which the tongue or cheek had been bitten, and now the frothy viscid saliva which filled the mouth to overflowing was deeply crimsoned with blood—a ghastly addition to a countenance already overcharged with horrors. During the last six hours the paroxysms have been less frequent and severe, but the vital powers are evidently fast ebbing away. "I cannot get my breath," was the answer slowly and almost inarticulately given to the question, "Have you much pain?"

Death happened about midnight, an hour after I had taken my leave, after a paroxysm of opisthotonos of no special violence, brought on, as it would seem, by an attempt to wipe away the bloody saliva from the lips. When I left the mind was perfectly clear and collected, and at no time, either before or after, was it otherwise.

For the rest it only remains to add (for the objections made to a post-mortem examination were insuperable) that the countenance appears to have retained after death the aged expression it had before death, and that the corpse when "laid out" was found to have stiffened without losing altogether the opisthotonic attitude. The mother of the patient is my only authority upon these points, for unfortunately it did not occur to me to make inquiries respecting them before the funeral had taken place.

In order to realize the points of resemblance and difference between this case and other cases of the kind, the course I propose to pursue is to take one after the other, as the points demanding attention, these—permanent muscular contraction, beginning by causing trismus, ending by causing opisthotonos, and implicating when at its height almost all the voluntary muscles except those of the hands, the eyeball, and the tongue; pain at the pit of the stomach, piercing through to the back; difficulty of swallowing, from the occurrence of spasm; fits of painful spasm in the permanently contracted muscles; risus sardonicus, and an aged expression of countenance; apnœa in the fits of spasm, and more or less dyspnœa at other times; increased temperature, without true fever; increased reflex excitability; absence of sleep; absence of numbness or tingling; absence of "head symptoms;" no marked want of control over the bladder and bowels; comparative voicelessness; the mouth clogged with viscid frothy saliva; a bitten tongue or cheek; dilatation of pupils; absence of priapism; presence of a wound; death by apnœa; early if not immediate rigor mortis.

Permanent muscular rigidity, causing, first, trismus, then opisthotonos, and implicating, when at its height, almost all the voluntary muscles except those of the hands, the eyeball, and the tongue.—Muscular rigidity, continuing without any marked relaxation from the time of its first appearance, is the most characteristic symptom of tetanus. It would seem to be the rule for this state of stiffness to begin in the muscles of the jaws, causing trismus, and to extend from thence as a centre, first to the muscles of the face and neck, then to those of the back, causing opisthotonos, then to those of the lower extremities, and, lastly, to those of the upper extremities, the progress in both extremities being from above downwards; but there are exceptions to this rule, for a few cases are on record in which the muscles of the neck have been affected before those of the jaws, and others, also only few in number, where the muscles near a wound, as of a stump after amputation, have been the first to become rigid. Even in the most extreme cases, the hands and the tongue are found to remain limper, and it is but very rarely, except perhaps in children with "head symptoms" in addition to the ordinary phenomena of tetanus, that a squint or a fixed stare shows that the deep muscles of the orbit are affected. Fits of spasm, of which more will have to be said presently, may seize upon the tongue, as they do very frequently upon the muscles of the throat in attempts to swallow, but there is no proof that either the tongue or the muscles of the throat are ever in a state of permanent contraction. Neither is there any certain proof that the heart or other involuntary muscles are in any degree permanently contracted. The affected muscles are very hard, curiously so, feeling very much as they do in rigor mortis, and they are not unfrequently somewhat tender when pressed or squeezed. In the great majority of cases, without question, the first effect of tetanic rigidity is to close the jaws and cause trismus, and the next to bend the body backwards and produce opisthotonos. Opisthotonos, indeed, is almost as characteristic and constant a phenomenon as trismus. Now and then, it is true, instead of the body being bent backwards it may be bent forwards (emprosthotonos), or sideways (pleurosthotonos), but these cases are quite exceptional, and *opisthotonos* may in reality be looked upon as the position which the body always takes or tends to take in tetanus.

Pain at the pit of the stomach piercing through to the back.—This is reckoned by the late Dr. Chambers as the pathognomic symptom of tetanus, and in fact it is scarcely ever absent, not even at the very beginning. This pain is especially severe in the fits of spasm, and then it is often agonizing, but it is present also, if not in a severe, at least in a mitigated form, in the intervals between these fits, scarcely ever ceasing altogether, even for a moment, when once it has made its appearance. It depends, there is little reason to doubt, upon the diaphragm being implicated in the tetanic condition. Once it was looked upon as a certain death-warrant, but this opinion, as Mr. Curling has shown, is untenable.

Difficulty of swallowing from the occurrence of spasm.—This spasm, which is provoked by the attempt to swallow, may be in the pharynx or gullet, or in the cardiac aperture of the diaphragm, one or all, making swallowing impossible, and often leading to the violent ejection of fluids through the nose or from the mouth. The distress consequent upon it may sometimes cause a horror of liquids not unlike that which exists in hydrophobia, and it always constitutes a grave difficulty, for it not only incapacitates the patient from feeding in the usual way, but it prevents him from being fed by means of the stomach-pump.

Fits of painful spasm in the permanently contracted muscles.—These fits become more frequent as well as more violent and painful as the disease progresses, recurring when at the worst every ten or fifteen minutes, and lasting from one to two and a half minutes. So violent has been the muscular contraction in some of these fits, that the teeth and thigh-bones have been broken, and great muscles like the psoas and recti femorales torn across. These fits of spasm are almost invariably very painful, the pain being that of cramp, but now and then the pain has been absent: thus, Sir Gilbert Blane mentions, on the authority of a surgeon in the navy, a case of severe tetanus, fatal in four days, in which the fits of spasm only gave rise to a sort of pleasurable tingling; and Mr. Curling instances an analogous case. Most generally the pain in the fit of spasm is felt chiefly at the pit of the stomach, and very often the pain in this region may be so agonizing and stifling as to make the patient insensible to pain elsewhere. Sometimes the pain in the neighbourhood of a wound, as in the stump after amputation, is that which is most complained of.

Risus sardonicus and an aged expression of countenance.—The sneering expression, caused by the angles of the mouth being drawn backwards and upwards, and known as the risus sardonicus, in association with spread nostrils, staring and prominent eyes, knitting of the brows, and bristling of the hair, is so often present as to be properly reckoned as pathognomic of tetanus. In the fits of spasm the lips are often drawn apart so as to expose the set teeth, but sometimes they are kept tightly pressed together by the spasmodic action of the orbicularis oris. The aged expression which was present in the case I have given, is exceptional, but it has been met with in other cases. Thus, Mr. Curling refers to a case of idiopathic tetanus, related by Dr. W. Farr, in which the patient, who was only twenty-six years of age, looked at least sixty; and he says further that he himself has "observed the same circumstance in an equally remarkable degree."

Dyspnœa with fits of comparative apnœa.—When tetanus is fully developed, an apprehension of suffocation is often present even in the intervals between the fits of spasm, and in these fits the suffused eyes, the livid countenance, and the agonizing struggle for breath show plainly enough that this is in no sense a groundless fear. How this difficulty is brought about is not easy to say, and probably the way is not always the same. Sometimes spasmodic closure of the glottis would seem to be a prominent cause; sometimes the thorax is, as it were, held in a vice by the spasm of all its muscles generally; most commonly perhaps these two causes act together. From my own small experience I should be disposed to attach more importance to the last cause than to the first, and I question whether much relief would be obtained in any case by carrying out Marshall Hall's suggestion of opening the windpipe in cases of tetanus.

Increased reflex excitability.—In P. M., as the disease advanced, the fits of spasm were brought on by the most trivial causes—a draught of air, a sudden noise, an attempt to swallow, an attempt to administer an injection, the arrangement of the bed-clothes, the lightest touch even—and hence it may be inferred that increased reflex excitability was an element in this case. Nor is this case at all exceptional in this respect. As the disease advances, in fact, the controlling influence of the nervous system is removed, and this is all, for what are counted as signs of increased reflex excitability are in reality no more than signs of nervous exhaustion, such as manifest themselves whenever the vital powers are sufficiently lowered by loss of blood, or in any other way.

Greatly increased temperature, without true fever.—In the fits of spasm, and in a lesser degree in the intervals between the fits, the skin is very hot and damp, this heat rising in some cases as high as 110·75° Fahr., the sweat having now and then a peculiar pungent smell. Usually the skin is literally drenched in perspiration and covered with sudamina. Usually the pulse is quick and weak; and if in the fits of spasm it acquires more force, the state of semi-suffocation then present shows very plainly that this change in its character is, as I have shown elsewhere, due, not to the injection of more red blood into the artery, but to the greater resistance which imperfectly aërated blood has to encounter in getting out of the artery.

The increased heat of skin in tetanus at first sight appears to show that fever is a part of tetanus, but further inquiry points to a very different conclusion. As death approaches, the temperature, instead of falling, as it might be expected to do, may actually rise higher, and, what is stranger still, the rise may not be at its maximum until the patient has been dead for some time. Dr. Wunderlich[1] gives three cases which establish this fact—which he was the first to observe—beyond all contradiction.

The first of these cases is one of idiopathic or rheumatic tetanus, the patient being a butcher, aged 29. The disorder, which presented nothing remarkable in its symptoms, ran its course in five days. Shortly preceding death there was some delirium, with marked abatement in the spasms, death happening in the exhaustion following a bout of spasm of no special severity. Putrefaction was unusually rapid. The brain was healthy, the cord was injected, and its texture (neuroglia) considerably broken down. The temperature of the ward at the time of death was 77° Fahr. What is of interest in the state of the breathing, the pulse, and the temperature, is as follows:—

Date.	Respirations.	Pulse.	Temperature. Degrees Fahr.
24th July, 1861	24	96	102
25th ,,	22	82	102
26th ,, 9 A.M.	20	96	104·45
,, ,, 6 P.M.	32	112	103·55
,, ,, 9.20 P.M.	36	180	110·1
,, ,, 9.35 P.M. *death*	...	...	112·55
After death 2′	...	...	112·77
,, 5′	...	...	113
,, 20′	...	...	113·22
,, 35′	...	...	113·55
,, 55′	...	...	113·67
,, 60′	...	...	113·55
,, 70′	...	...	113·22
,, 90′	...	...	113
,, 100′	...	...	111·8
,, 6 hours	...	...	106·25
,, 9 ,,	...	...	104
,, 12 ,,	...	...	102
,, 13½ ,,	...	...	101

The second case is one of traumatic tetanus in a man aged 20, fatal tenth day. Up to twenty-four hours before death the tetanic symptoms were fully marked, and the mind quite clear; at this time, and especially in the six hours immediately preceding death, unrest, talkativeness, jactitations, and slight delirium were more prominent symptoms than the spasms. The appearances after death were like those found in the first case. In the last three days the mercury went up slowly and steadily from 100° to 105°·8, at which point it stood three hours before death; at death and afterwards the notes made of the temperature are these:—

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[1] Archiv der Heilkunde, Bd. ii., iii. and v. (1861, 1862, and 1863).

At death	107°·6 Fahr.
10′ after death	107·8
15′ ,,	108·
20′ ,,	107·8
38′ ,,	106·45
58′ ,,	105·8
1 hour 5′ ,,	105·35
,, 20′ ,,	104·45
,, 35′ ,,	103·55
2 hours ,,	101·75
4 ,, ,,	99·3

The third case is that of a man, aged 57, a bookbinder by trade, with idiopathic or rheumatic tetanus. Tetanic symptoms set in in the usual way on the 20th June, 1863, and were fully developed two days afterwards, when also symptoms of pneumonia were detected. Death happened towards the end of the day following, as much or more from the pneumonia as from the tetanus; and after death the only very marked appearances were those of pneumonia. In this case the temperature, which was never higher than 104°·55, was—

3½ hours before death.	102°·85 Fahr.
At death	not stated
10′ after death	103·32
21′ ,,	103·55

Other cases are also on record which show that this strange rise in temperature up to death and after it, is not peculiar to tetanus. Dr. Wunderlich gives three such cases:—(1) A case apparently of lead-poisoning in a plumber, ending fatally in 40 hours: the symptoms being sudden insensibility, and, later on, tetanic and epileptiform convulsions. In this case the temperature at death was 107°·7, and there was some slight increase afterwards; (2) a case of cerebro-spinal meningitis, with unrest, delirium, and retraction of head for four days, and then sopor, in which the temperature was 107°·26 at death and 107°·37 after death; and (3) a case of rheumatic fever, with cerebral meningitis, shortly ending in coma on the sixth day, in which the temperature ranged from 109°·62 to 110°·75 in the five hours before death, and stood at 111°·87 thirty minutes after death. Dr. Erb[1] also gives three cases with fuller detail, namely, these:—

A young man, aged 22, with tubercular inflammation of the base of the brain, who died without convulsions, after having been in a state of profuse perspiration and unconsciousness for twenty-four hours, with rapid respiration (60 to 44), and a pulse quite uncountable towards the end. In this case the temperature in the twenty-four hours before death, at death, and afterwards, was as follows:—

24 hours before death	from 102°·65 to 104°·9 Fahr.
At death	104·9
13′ after death	105·12
25′ ,,	104·67
55′ ,,	104

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[1] Deutsches Archiv für Klin. Medicin. vol i. 1866.

A woman, aged 22, six months gone in pregnancy, who died with symptoms of coma, without convulsions, the coma having set in suddenly an hour and a half before death, and in whom signs of purulent meningitis were detected after death. During the comatose state the breathing was very laboured, and the pulse full and frequent. The temperature, of which the notes are as follows, was only taken after death:—

At death	Temp. not stated.
6′ after death	103·45 Fahr.
10′ ,,	104
15′ ,,	104·67
20′ ,,	104·9
25′ ,,	104·9
35′ ,,	105·12
45′ ,,	105·12
1 hour 40′ after death	104
2 hours 40′ ,,	101·22

A woman, aged 22, who after having suffered from diabetes mellitus in its ordinary form for three and a half years, passed into a state of sopor, after having had headache and some delirium for twenty-four hours. Death happened in about forty-eight hours from the commencement of the head symptoms. During the twenty-four hours of sopor preceding death, the degree of heat ranged from 102°·65 to 106°; at death and afterwards the notes made of the temperature are these:—

At death	106° Fahr.
5′ after death	106·25
15′ ,,	106
25′ ,,	105

The body has been found to become very hot before death, and to remain very hot after death, in cholera, in yellow fever, and in several other cases, of which instances are given by Dr. Erb and by several other writers in Germany, and by Drs. Ringer,[1] Weber,[2] Murchison,[3] Sanderson,[4] and others in this country; the cause of death in the majority of these cases being some sudden affection of the brain, coma in others; but there are but few cases in which the heat of the body has been found to rise after death. Indeed, I know of no such cases besides these I have quoted, except one, which came under my notice a short time ago—a case of a man, aged 60, who died from sunstroke in twenty-six hours, the symptoms being sudden coma, with great oppression of the pulse and breathing, without convulsion. In this case the temperature was—

12 hours before death	103°·25 Fahr.
3 ,, ,,	104
At death	not ascertained.
7 hours after death	105·5

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[1] Med. Times and Gazette, vol. ii. 1867.

[2] Clinical Soc. Trans. vol. i. 1868.

[3] Ibid.

[4] Ibid.

If, then, the temperature rises in this manner under these circumstances, it is more than difficult to connect the increased heat of tetanus with increased activity of the circulation or with anything like fever in the ordinary form of the word. The temperature rises as the time of death approaches, when the state of the circulation must every moment be becoming more and more the reverse of increased activity: the temperature continues to rise even after actual death, when the blood has come to a standstill. These are the facts; and, these being the facts, it may be that the increased heat in tetanus may be connected, not with increased activity of the circulation, not with true fever, but rather with the contrary state of things. Nor is it more easy to connect the increased heat of tetanus with the spasms. A part of the increase may be accounted for in this manner, but only a small part. Indeed, the simple fact that in one of the cases which has been instanced a marked abatement in the severity of the spasms was accompanied by an actual rise in the column of mercury, and that the column continued to rise after death, when all spasm is at an end, is in itself a sufficient proof that it is not in muscular action that the explanation of the increased temperature of tetanus is to be found. Moreover, the fact that the temperature rises in the same way before and after death, in cases where neither convulsion nor spasm was among the symptoms during life, must lead to the same conclusion. How to explain the increased heat of tetanus is another matter. Increased heat, as was shown in the preliminary remarks, is an effect of injuries by which the cord or medulla oblongata is torn or cut across. Increased heat, as is seen in some of the cases which have just been alluded to, is an accompaniment of certain diseases which annihilate, more or less completely, cerebral action. It seems as if one condition of this change in temperature was the paralysing of a regulating cerebral influence; and beyond this it is difficult to see further, except it be that this paralysis, reaching to the vaso-motor nerves, allows the minute vessels to dilate and receive more blood, and that the increased quantity of blood, even though this blood be stagnant, may lead to increased molecular changes, of which increased heat is an effect. What is necessary, however, is not to find the cause of the increased heat in tetanus, but simply to point out the fact that increased heat in this case does not imply increased activity of circulation—that true fever, in the ordinary sense of the word, is not a part of tetanus. And this, as it seems to me, is a legitimate inference from the evidence which has been cited, and the comments which have been made.

Absence of sleep.—In the acute cases, sleep, as a rule, is banished altogether, and even in the subacute cases this blessing is only realized in unrefreshing broken snatches. Want of sleep, indeed, is one of the not least distressing features of this disease. "The muscles," says Mr. Curling, "are observed to be relaxed during sleep, a striking example of which occurred to Mr. Mayo in a boy who recovered from the disease. On visiting his patient before the symptoms were subdued, Mr. Mayo found him asleep, and remarked that he lay perfectly relaxed. The abdominal muscles were soft and yielding, and had not the least tension. The boy was awakened, and at the instant the full tension of the muscles returned. Not being further disturbed, he fell asleep in a few minutes, when the muscles again became relaxed, and again, on his being awakened, resumed the state of spasm. I have, on several occasions, witnessed the same phenomena." Except the biting of the tongue, on waking from a brief nap, be a reason for believing that the muscles of the jaws had been relaxed during sleep, so as to allow the tongue to get between the teeth, there was no proof that the muscles were relaxed during sleep in the case I have given; but in other cases I have had proof sufficient of this relaxation.

Absence of numbness and tingling.—Of this there can be no doubt—that numbness and tingling form no part of the history of tetanus.

Absence of "head symptoms."—The mind is clear from the beginning to the end of the disease almost invariably, and not unfrequently it is a matter for wonder how well the patient bears up under his atrocious sufferings—a marked difference this between tetanus and hydrophobia. And in the few instances in which delirium or coma has made its appearance a short time before death, it is not improbable, as more than one writer has observed, that this derangement is often more the result of the remedies employed than of the disease.

No marked want of control over the bladder or bowel.—In tetanus there is, as a rule, none of the difficulty with the bladder which is almost invariably met with in acute spinal meningitis. The bladder may act seldom, but it is not incapable of acting. Constipation is a common but not a constant symptom, and when it is present it may be a question whether, like the "head symptoms," it is not as much due to the medicines used as to the disease. Now and then, however, there may be great difficulty in voiding the contents of the bladder and bowels, and in some of these cases the resistance to the introduction of a catheter or enema-pipe has shown that a part of this difficulty is owing to spasm of the compressor urethræ or sphincter ani.

Comparative voicelessness.—This phenomenon is readily accounted for as a result of the spasmodic interference with the action of the chest and of the tight shutting of the jaws. Indeed, it could not well be otherwise in the fully developed disease.

The mouth clogged with viscid frothy saliva.—This is a common if not a constant symptom, though not so marked in degree as in hydrophobia, and there is no difficulty in accounting for it in either case, for the inability to drink and swallow will explain at one and the same time why the saliva is viscid and why it accumulates in the mouth.

A bitten tongue or cheek.—This accident is of rare occurrence, and its rarity may be taken as an incidental proof of sleeplessness as a symptom of tetanus, for it is to be supposed that the opening of the jaws, from the relaxation of their muscles during sleep, would allow the tongue or cheek to get between the teeth—to get into that position in which the spasm which attends the moment of waking would be sure to crush it.

Dilatation of pupil.—This condition was always present in the case which serves as my text, especially in the fits of spasm, and this has been the rule in several cases of tetanus in which I have examined the pupil. Mr. Curling, on the contrary, found the pupil contracted in the majority of his cases.

Absence of priapism.—Mr. Morgan states that priapism occurs occasionally; but this observation is not confirmed by other writers on the subject. I have never seen it, and I am very much disposed to think that the case or cases in which Mr. Morgan saw it were cases, not of tetanus, but of acute spinal meningitis, in which disorder priapism is an occasional symptom.

Presence of a wound.—The great majority of cases of acute tetanus appear to be in some way dependent upon a wound or injury of one kind or another in one place or another. I shall have occasion to refer to this relationship elsewhere: and at present I would only notice, in passing, the presence of a wound which, to all appearance, presented no indications of an inflammatory or otherwise unhealthy character.

Death by apnœa.—Apnœa is one way, and perhaps the common way, in which death is brought about in tetanus. Not unfrequently, however, the patient sinks from asthenia, having been to a great degree free from fits of suffocative spasm for some time before death. Spasm of the heart has also been mentioned as a method of dying in tetanus, and the heart has not unfrequently been found to be curiously hard and contracted after death; but an examination of the facts tends very much to discountenance this idea, and to show that death is either by apnœa or asthenia, singly or together.

The immediate occurrence of rigor mortis.—Sommer and others have noticed that rigor mortis may occur without any appreciable interval of muscular relaxation after death from convulsions, and Dr. Brown-Séquard has confirmed this observation and given a definiteness to it which it had not before. He has indeed done more than this, for he has not only confirmed the fact that rigor mortis may occur without any appreciable interval of muscular relaxation, but he has established the law that rigor mortis is long in coming on and long in passing off where death was not preceded by any long-continued violent action of the muscles, and that it is quick in coming on and quick in passing off in direct proportion to the amount of long-continued violent action which preceded death. In many animals killed by strychnine, for example, in which death was brought about, not by one violent spasm, but by many, he has found rigor mortis set up before the heart had ceased to beat. Nay, he even refers to the case of a man under his own observation in which rigor mortis occurred before the heart had ceased to beat. I have never witnessed this phenomenon either in animals or in man; but I have more than once failed to find any line of separation between tetanic stiffness and cadaveric rigidity in animals killed by strychnine, or by the shocks of a Ruhmkorff coil: and I am therefore quite prepared to understand that in P. M——'s case, where there were many convulsions before death, rigor mortis may have occurred without any appreciable interval of muscular relaxation, and in this way fixed in the corpse the aged expression of the countenance, and the opisthotonic attitude.

Two distinct varieties of tetanus are usually recognised, and properly so,—the *traumatic*, in which a hurt of some kind or other is believed to be the primary cause; and the *idiopathic*, in which the only obvious cause would seem to be exposure to cold and damp. In each variety the symptoms are much the same, any difference of moment being only one of degree. In the acute form, the spasms come on suddenly, occur frequently, and grow in violence with each recurrence; in the less acute forms the spasms are more slowly developed in the first instance, the paroxysms are comparatively far between, and they do not recur with increasing rapidity and violence. The traumatic, as a rule, is more acute than the idiopathic variety. Trismus nascentium is considered by many as a distinct variety of tetanus, but this appears to be a distinction without a real difference. It is tetanus in newly-born infants,—traumatic, because the wound of the navel seems to have a good deal to do with its production, and at the same time idiopathic, for it is certain that cold and damp, and foul air, and other general causes also figure conspicuously as sources. It is, indeed, to this form of tetanus that a remark of Sir Thomas Watson applies especially, which is applicable to all forms, namely this, that "although tetanus may be excited by a wound, independently from exposure to cold, or by cold, without any bodily injury, there is good reason for thinking that, in many instances, one of these causes alone would fail to produce it, while both together call it forth."

2. Post-mortem Appearances.—There are no morbid changes in the nervous system peculiar to tetanus. "Serous effusion with increased vascularity," says Mr. Curling, "is generally observed in the membranes investing the medulla spinalis, and also a turgid state of the blood-vessels about the origin of the nerves," and the same changes may also be met with in the cranium, but in a less degree, and less frequently. It is also a fact of considerable moment in relation to this point, that Majendie, Ollivier, and Orfila failed to detect any perceptible lesion in the spinal cords of animals dying from the tetanus produced by strychnia. Out of seventy fatal cases collected by Mr. Curling, there were only two in which changes in the nervous system unequivocally the result of inflammatory action were discovered after death, and these two were cases where there had been a blow or wound to the back, where the symptoms had plainly to do with the inflammation of the cord or its membranes rather than with tetanus, and where the signs of inflammation found after death may, to say the least, be referred to the injury quite as easily as to the tetanus. Mr. Curling also

points out, as a fact not to be overlooked, that the turgid state of the vessels of the pia mater, together with the effusion of serum which is met with in the spinal cord and brain after death from tetanus, is also met with in those persons who may have been poisoned by opium, hydrocyanic acid, and other powerful agents often employed in the treatment of tetanus, as well as after death from delirium tremens, hydrophobia, epilepsy, and other diseases; and, as bearing upon these exceptional cases, in which unequivocal signs of inflammation in the cord or brain have been met with after death from tetanus, he says, "Whether inflammation be the result of injury or arises spontaneously, it is worthy of notice that the spasms, though continued and severe, do not occur in such violent paroxysms as in traumatic tetanus." Neither can the preternaturally injected state of the minute vessels supplying the sympathetic ganglia, especially the cervical and semilunar, met with by Mr. Swan and others in some cases of tetanus, be looked upon as at all constant phenomena after death from tetanus.

Nor do recent microscopic investigations into the condition of the spinal cord in tetanus bring to light any clearer signs of inflammatory change. Mr. Lockhart Clarke[1] finds the vessels injected, and the substance of the cord in a state varying from simple softening to complete solution, the softened or dissolved portions forming irregular "areas of disintegration," filled with the *débris* of blood-vessels and nerves, or with a fluid finely granular or perfectly pellucid. These areas of disintegration were chiefly in the grey substance around the canal, but they were also in the white substance. They were, in fact, in no one part exclusively or particularly. Here and there were extravasations of blood, and "other exudations," but pus corpuscles are not mentioned. "In the walls of the blood-vessels," Mr. Clarke says, "there was no morbid deposit, nor any appreciable alteration of structure, except where they shared in the disintegration of the part to which they belonged; but the arteries were frequently dilated at short intervals, and in many places were seen to be surrounded, sometimes to a depth equal to double their diameter, by granular and other exudations, beyond and amongst which the nerve-tissue, to a greater or less extent, had suffered disintegration." "The appearances met with," says Mr. Clarke, are "exactly similar in kind to the lesions or disintegrations which I find in various cases of ordinary paralysis, in which there is little or no spasmodic movement." The cord is broken up, in fact, as at a certain time it is broken up by ordinary putrefaction, and, the dilated vessels and certain exudations of blood and serum excepted, this is all that is noticed. The case points to disintegration, not to inflammation; and what Mr. Clarke finds in six cases is substantially the same as that which Dr. Dickenson[2] finds in the one case examined by him. Indeed the only peculiarity in this latter case is, in the presence, in addition, of an excessive quantity of a translucent, structureless, and finely granular, carmine-absorbing material, evidently the sero-fibrinous plasma of the blood, which has escaped from the minute arteries into various parts of the substance of the cord where the nerve-tissue has broken down, or which lies in pools here and there between the cord and its membranes. It is a state of œdema rather than anything else, certainly not a state of inflammation.

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[1] Med.-Chir. Transactions, vol. xlviii. 1865.

[2] Ibid. vol. li. 1868.

Traces of inflammation in the wound, especially in the injured nerves, may be met with after death from tetanus, and more frequently than in the spinal cord or other great nervous centres; but these again, instead of being constant, are not even common appearances. In the great majority of cases, indeed, the wound, if there be one, is perfectly healthy and healing. Neither are there any other post-mortem facts which can be looked upon as essential to tetanus, for those which remain to be mentioned, as ruptured muscles, broken or dislocated bones, engorged lungs, injection and contraction of the pharynx and palate, worms in the alimentary canal, and others, are plainly accidental and exceptional.

3. Causes.—The two great causes of tetanus are, as has been mentioned already, cold and damp, and bodily injury of some sort. Exposure to cold and damp tells most in this manner when acting upon a body previously relaxed by heat and perspiring, and this is all that can be said, except that this exposure is more likely to issue in tetanus in a foul atmosphere than in a fresh one. As regards the hurt which may give rise to tetanus, it is difficult to know what to say. In the Peninsular war, as Sir James McGregor states, tetanus supervened on every description and in every stage of the wounds, from the slightest to the most formidable, in the healthy and sloughing, the incised and lacerated, the most simple and the most complicated; and this statement expresses the opinion of all surgeons, military and others. Indeed, all that can be said is, that punctured wounds seem to be more likely to issue in tetanus than incised, and wounds in the extremities more than wounds in the head, breast, and neck. And certainly an inflammatory condition of the wound cannot be regarded as essential. In a great number of cases, in the majority perhaps, the primary wound was completely healed and almost forgotten when the symptoms of tetanus made their appearance; and Dr. Rush, who had extensive opportunities for observation in the military hospitals of the United States, and who was unquestionably a most competent observer, remarks that there was invariably an absence of inflammation in the wounds causing the disease. John Hunter also says: "The wounds producing tetanus are either considerable or slight. . . . When I have seen it from the first, it was after the inflammatory stage, and when good suppuration was come on; in some cases when it had nearly healed, and the patient was considered healthy. Some have had locked jaw after the healing was completed. In such I have supposed the inflammation to be the predisponent cause, rendering the nervous system irritable as soon as it was

removed. When tetanus comes on in horses, as after docking, it is after the wound has suppurated and begun to heal." There is, indeed, abundant evidence to show that an inflammatory condition of the wound is not necessary to the production of tetanus, and some evidence even which is calculated to lead to a contrary conclusion, by showing that where an inflammatory condition of the wound has been present, this condition has passed off before the tetanic symptoms made their appearance—the inflammation, to repeat the words of John Hunter just used, "rendering the nervous system irritable *as soon as it was removed*," not rendering it irritable as long as it was present. The interval between the hurt and the development of the tetanic symptoms varies considerably. In eighty-one of the cases collected by Mr. Curling, the symptoms made their appearance between the fourth and fourteenth days, both inclusive, and in nineteen on the tenth day. Four cases are also given in which the symptoms came on more speedily, one (somewhat doubtful) almost instantaneously, another in one hour, a third in two hours, and the fourth in eleven hours, and, at the other extreme, one in which they were deferred as late as the tenth week. In traumatic tetanus the sooner the symptoms show themselves the more acute and dangerous is the malady. In idiopathic tetanus the symptoms, as a rule, commence sooner than in traumatic tetanus, often in a few hours; but the idiopathic, notwithstanding, is generally of a more chronic kind than the traumatic, and far less dangerous.

Tetanus is not a malady peculiar to any country, or climate, or people, but it is more common in hot countries than in cold. It would appear, also, that negroes are more likely to be attacked than whites. Great atmospheric changes, especially from heat to cold and damp, as to a cold and dewy night after a sultry day, are evidently most favourable to the development of tetanus, and so in a less degree are foul air, despondency, terror, physical exhaustion. It must be confessed, however, that cases of idiopathic tetanus, as compared with those which are traumatic, or partly idiopathic and partly traumatic, are, to say the least, extremely rare in this country.

4. DIAGNOSIS.—The differences between tetanus and acute spinal meningitis are sufficiently marked to prevent any confusion as to diagnosis if only a moderate degree of attention be paid to the subject. In tetanus the jaw is firmly set from the first, and, in addition to the fits of spasm, there is permanent muscular rigidity between the fits: in spinal meningitis, if the jaw be set at all, it is rather at the close of the disease, and then only in an inconsiderable degree, and spasms or muscular rigidity are neither constant nor conspicuous phenomena. In spinal meningitis, indeed, it is plain that the muscular rigidity and seeming spasms are in great measure voluntary or semi-voluntary acts to prevent the pain in the back and limbs which is produced by movement, and that the muscles are relaxed almost as long as the patient can keep perfectly still. In a word, the true involuntary fits of spasm

and the permanent muscular rigidity which are constant and characteristic phenomena in tetanus, are not present in acute spinal meningitis.

Nor can hydrophobia be very well confounded with tetanus. In tetanus the features are drawn into the risus sardonicus, the eyes are natural, and the whole countenance is expressive of pain and suffering, —nothing more: in hydrophobia there is an impress of excitement and distress and horror and unrest upon the features which has no counterpart in the tetanic countenance. In tetanus the body is for the most part rigidly fixed in one position by tonic spasm; in hydrophobia the spasmodic movements are clonic, and the body is in a state of perpetual unrest until the stage of final exhaustion. In hydrophobia, noisy attempts are continually made to spit and hawk away the viscid phlegm which clogs the mouth and throat—the noises being sometimes not altogether unlike the bark of a dog—and any effort to relieve the tormenting thirst, or even the bare thought of such an effort, brings on the fit of fear and convulsive agitation which has given rise to the name hydrophobia: in tetanus there are no symptoms which can be considered as strictly comparable to these. In tetanus, finally, the mind is clear to the last, whereas in hydrophobia there is almost from the first a peculiar and often very wild delirium.

The tetanic symptoms produced by strychnia and some other poisons may be more easily confounded with traumatic tetanus, but even here it is possible, with care, to make a correct diagnosis. It is possible, as Dr. Christison pointed out, for strychnia to be given in repeated doses so regulated as to produce a train of symptoms scarcely, if at all, distinguishable from traumatic tetanus; but not so if, as is usually the case, an amount sufficient to produce death be given in one dose. In this latter case, indeed, the differences of the symptoms are sufficiently marked. In the toxic tetanus the symptoms run a rapidly fatal course, death happening in a quarter of an hour, half an hour, and usually within the hour: in traumatic tetanus, with very few exceptions, life is prolonged for two or three days at least. In the toxic tetanus the arms are stretched stiffly out, the hands clenched, and the legs separated widely from each other and rigidly extended: in traumatic tetanus the hands are usually free from spasm, and the arms nearly so, and even the legs are scarcely ever affected to the degree which is seen in toxic tetanus. In the tetanus caused by strychnia, Mr. Poland says, "The patient can open his mouth to swallow; there is no locked jaw:" in traumatic tetanus, locked jaw is the first and most constant manifestation of the spasm.

The jaw may be locked for a long time, and various muscles in other parts may be affected with continuous spasm in cases in which hysteria is supposed to figure largely as a cause—cases in which there is the condition called spinal irritation: but these cases, as will appear in due time even when most like, are in reality so unlike tetanus as scarcely to deserve even this passing mention.

5. PROGNOSIS.—In the cases "in which the access is slow, the spasms by no means violent, the paroxysms slight and recurring at long intervals, and where the patient can obtain sleep, whether traumatic or not, we may generally anticipate a favourable result;" and, again, "the longer the interval before the appearance of the symptoms, the more chronic the disease, and the greater the probability of recovery." So speaks Mr. Curling of the chronic cases of tetanus in contradistinction to the acute; and in illustration of the probability of recovery, he adds: "In thirteen cases, symptoms of tetanus occurred about three weeks after the wound, and four only were fatal; and of seven cases in which they did not make their appearance till after a month, only two ended fatally." In the cases, on the other hand, in which the spasms supervene rapidly upon the injury, and recur with increasing violence at decreasing intervals, and in which sleep is banished, a vast majority die—die, as Hippocrates noticed ages ago, within four days. Death may happen in a fit of suffocation in which sometimes there is obviously spasm of the glottis, but more frequently it would seem to be brought about by asthenia after a fit of spasm. The time occupied in recovery varies greatly,—one, two, three, four, five, six, seven, eight weeks, or even longer. A certain degree of weakness and stiffness may also remain in the muscles long after recovery. In one case rigidity of the muscles of the jaw remained for six months; in another it returned whenever the patient caught cold up to nine months; and in a third, at the end of three years, it is stated that the "features retained the indelible impression of the disease." These cases are given by Mr. Curling.

6. TREATMENT.—After passing in review the principal remedies that have been tried in tetanus—opium, blood-letting, the cold bath and cold affusion, ice to the spine, the warm bath, bark, wine and spirits, mercury, purgatives, foxglove, tobacco, musk, prussic acid, carbonate of iron, oil of turpentine, strychnia, woorali, ether and chloroform inhalations, amputation, division of nerves, tourniquets—Sir Thomas Watson says: "In all cases, there being no special indication to the contrary, I should be more disposed to administer wine in large quantities, and nutriment, than any particular drug;" and this statement, I take it, expresses a very general feeling in this country. For my own part, I should certainly be more disposed to trust to alcohol than to any drug; but, in saying this, I do not say that I should place no confidence in drugs. I should certainly place no confidence in any sedative or narcotic given by the stomach in sedative or narcotic doses; but, on empirical as well as on theoretical grounds, I should say that opium can scarcely be dispensed with, and that chloroform or ether inhalations will be of infinite service in relieving pain and spasm, and that too without compromising the chances of recovery, if care be taken to pour in wine and to supply nourishment at the same time so as to prevent the patient from waking up almost immediately after the inhalation.

If the *rationale* of spasm be that which is hinted at in the preliminary remarks, the great indication of treatment must be, not to depress the circulation, but to rouse it into greater activity; and one reason why the treatment of tetanus has been so eminently unsatisfactory may be that this indication has not been fully realized and carried out. In tetanus much wine may be given without producing anything like intoxication, or without relaxing the spasms in any degree. The system in this disease is altogether insensible to the action of wine in ordinary doses. As to this there can be no doubt. Whether a different result would have been arrived at if alcohol had been given more boldly, ardent spirits in place of wine, ardent spirits undiluted rather than diluted, is yet an open question, but I am disposed to think that the spasms might have been conquered without compromising the safety of the patient if this had been done. There are now not a few cases on record which show that the bite of a rattlesnake or cobra or other deadly serpent may be prevented from killing by at once giving ardent spirits in sufficient quantity, and I am disposed to think that these facts have an important bearing upon the treatment of tetanus. There are, undoubtedly, great differences between the condition in tetanus and the condition in these poisoned bites, but there are also certain resemblances which must not be lost sight of. There is the same insensibility to the action of alcohol in ordinary doses; there is an exhaustion to be counteracted, which is more rapidly fatal in the poisoned bite than in tetanus, but which in acute tetanus is sufficiently rapid to create the gravest fears, and to justify the most heroic measures; there may even be a poison at work in both cases as well as a wound, a poison introduced into the wound in one case, a poison generated in the wound in the other case. There are resemblances between the two cases, indeed, which, though not very close, may be close enough to justify the hope that a practice which has been found to answer in the bite of a poisonous serpent may also be found to answer in acute tetanus.

In speaking thus, it is not intended to imply that ardent spirits are the only way of fulfilling what has been said to be the primary indication of treatment in tetanus. Eau de luce has been found to be of great service in the bites of serpents, and it might be of service in tetanus. Ether, also, might be of use, or turpentine, or camphor, or ammonia. But to my mind these and other medicines of a like nature are more likely to disorder the stomach and system generally, and in other respects are less manageable and less certain in their action, than ardent spirit.

As regards local measures it is less difficult to arrive at a conclusion. In many cases, no doubt, there is an eccentric irritation, starting from the wound or some other point, and much good would be done if this could be removed. It is probable, also, that this end might be gained in more ways than one, and that one very direct way is by the subcutaneous injection of various substances—morphia, atropine, woorali, conia (which seems to be strictly analogous in its action to woorali), Calabar bean, &c. The results of these injections in causing the relaxation of spasm in connexion with the minor forms of spinal irritation are very encouraging. One thing, however, ought to be borne in mind, and that is, that these injections should be used so as not to produce a general depressing or paralysing effect upon the nervous system. All that ought to be aimed at is to obviate local irritation merely; and, to my mind, to go beyond this point is both wrong in principle and dangerous in practice.

For the rest, it is, of course, desirable that the patient should be carefully guarded from cold, and from anything which would excite or disturb him, as too much light or noise, or too meddlesome nursing. In a word, quiet and warmth are not only desirable: they are indispensable.

V. Locomotor Ataxy.

Until very recently the disease which forms the subject of the present article was confounded with paraplegic diseases. The difficulty in locomotion, which is the most characteristic symptom, was supposed to be owing to simple paralytic weakness of the legs. It was not perceived that the legs, in the earlier stages of the disease at least, had lost little, if any, of their power to act separately—so little, indeed, that it might require all the force of a strong man to bend or straighten them against the will of the patient—and that what they had lost was that power of co-ordination by which the two limbs are enabled to act together, as they have to do in standing and moving about. The credit of having first drawn this distinction, and at the same time shown that this want of co-ordinating power is so associated with a definite group of other symptoms as to deserve to be regarded as a distinct disease, is due to Dr. Duchenne (of Boulogne). Before this time, no doubt, the characteristics of such a disease had been more or less clearly realized. They had been described, in fact, under the old name of *tabes dorsalis*, especially in the sketch of this disease given by Dr. Romberg. They had been detected by the late Dr. Todd, and not only so, but associated with that particular lesion with which they are now known to be connected, namely, with chronic disease of the posterior columns of the spinal cord. "Two kinds of paralysis of motion," wrote Dr. Todd, "may be noticed in the lower extremities,—the one consisting simply in the impairment or loss of voluntary motion; the other distinguished by a diminution or total loss of the power of co-ordinating movements. In the latter form, while considerable voluntary power remains, the patient finds great difficulty in walking, and his gait is so tottering and uncertain, that his centre of gravity is easily displaced. The cases are generally of the most chronic kind, and many of them go on from day to day without any increase of the disease, or improvement of their condition. In two examples of this variety of paralysis I ventured to predict disease of the posterior columns, the diagnosis being founded upon the views of the functions of the columns which I advocate; and this was found to exist on a post-mortem inspection; and in looking through the accounts of recorded cases, in which the posterior columns were the seat of lesion, all seem to have commenced by evincing more or less disturbance of the locomotive powers." (Cyclopædia of Anatomy and Phys. vol. iii. p. 721, S.)

Dr. Todd published these remarks in 1845; Dr. Duchenne's first memoir appeared in 1857. Dr. Todd must, therefore, have the credit of having anticipated Dr. Duchenne; but still the lion's share of honour must be assigned to the latter, for the plain fact is that Dr. Duchenne has developed in a series of formal memoirs what Dr. Todd has only indicated in these few sentences. In a word, it must be allowed that Dr. Duchenne deserves almost the entire credit of being the first to detect the exact features of the disease now known as progressive locomotor ataxy, and to call the attention of others to the subject.

The name of progressive locomotor ataxy (*ataxie locomotrice progressive*), from α, privative, and τάξις (order), is that which was chosen by Dr. Duchenne. It is not a very fortunate one, but it has been adopted, and must be retained, until a better one is found. It is certainly to be preferred to *tabes dorsalis*, for this name is commonly supposed to imply past incontinence on the part of the patient. How far it is right to perpetuate the cheerless affix *progressive* is, however, very questionable. At present, no doubt, the prognosis is full of gloom. From bad to worse is the common course of things, but, at the same time, there are cases—and their number is increasing every day—in which the symptoms have been long stationary, and others in which there has been unequivocal amendment. But even if the element of hope were wanting, it is surely desirable not to bring this unhappy fact into undue prominence. It is surely not necessary to continue to use an epithet of which the effect must be to frighten the patient and discourage the practitioner, and this too without compensating advantages of any kind. As it seems to me, indeed, everything is gained by the name locomotor ataxy which is gained by the name progressive locomotor ataxy; and nothing is lost but what can well be spared; and therefore in what I have to say I shall drop the term "progressive," and speak simply of "locomotor ataxy."

1. SYMPTOMS.—As a text for what I have to say upon the symptoms of locomotor ataxy, I take from my note-book a case which was some time ago (April 1865) under my care in the National Hospital for the Paralysed and Epileptic.

Case.—A sailor, thirty-four years of age, by name J. C——, well proportioned, unusually well developed as to muscle everywhere, very lean, and much bronzed by long exposure to sun and sea.

(*a*) This man is capable of walking without a stick, but his gait is peculiar—staggering, precipitate, the legs starting about vaguely and spasmodically, and the heels coming down heavily at each step. With his eyes shut, or in the dark, he reels over at once and falls to the ground, if left to himself. Sitting or lying down, he can lift either leg steadily into any position, and fix it there so firmly that it is out of my power to bend or straighten it against his will. In order to do this, however, he must see what he has to do, for if his eyes are shut, his limb at once becomes uncertain and unsteady in its movements, and comparatively powerless. His right leg is a little weaker than the left, but not in any well-marked degree. He finds it very difficult to come downstairs, or to turn round, or to quicken his pace much, and he is speedily fatigued by the acts of standing or walking. On being told to shut his eyes, and touch his nose with the forefinger of each hand in turn, he does so with tolerable accuracy, especially with the forefinger of the left hand. On being told to stretch out his arms, and keep them out, he does so, but only so long as he is allowed to see what he was doing; for, on holding a book up before his eyes, the arms, shoulders, neck, and head—the upper part of his body generally—at once became afflicted with convulsive agitation. When the book was taken away, these movements speedily came to an end, but not before they had issued in a fit of crying and sobbing which was not a little distressing to witness. This fit took the patient quite by surprise, and it could not be accounted for by the examination having been conducted roughly, or carried on for an undue length of time; indeed, the holding of the book before the eyes, which was the immediate cause of the fit, did not occupy more than a minute at the most.

The muscles of the lower limbs stand out firm and hard when made to contract by the will, and the contraction seems to be not at all wanting in force. Indeed, as has been already stated, it is out of my power to bend or extend the limb against the will of the patient. There is no tremulousness in the legs or elsewhere, and there are no marked reflex movements when the soles of the feet are tickled.

What is complained of chiefly are severe pangs of pain, stabbing, boring, in flashes like lightning, flitting from one spot to another in a very erratic manner, recurring in paroxysms varying in length from a few minutes to twelve, twenty-four, or forty-eight hours, and generally remaining at the same spot during the same paroxysm. These pangs are most frequently felt in the two feet, especially along the outer side of the metatarsal bone of the little toe; and they also are not unfrequently met with at the back of the thigh, in the nates, and in the upper arm about the lower part of the belly of the biceps. They are scarcely ever absent, especially at night; at night, too, there is often a sensation of great coldness, with some degree of constriction at the seat of pain.

Tactile sensibility, measured by the compasses, is found to be much impaired in both feet, especially in the soles, in the calves of both legs, and to some degree also at the back of the thighs, in the nates, and in the palms of the hands. The ground is felt very obscurely, but, so far as it is felt, the sensations are accurate—that is to say, it does not seem as if there were elastic cushions, pebbles, or other imaginary bodies upon the floor, or as if the feet had nothing under them but free air, as is sometimes the case. Very rough pinching is scarcely at all felt in the benumbed parts, but elsewhere the sensibility to painful impressions is keen enough.

There is also evident impairment of the proper sensibility of the muscles, joints, and bones in the limbs, and especially in the legs. Thus, the patient never knows clearly where his feet are without looking at them, and now and then he has been so uncertain in this respect, that a foot has slipped out of bed without his being the wiser; and thus, again, his finger has not the power of discriminating between a sovereign and a shilling by the weight merely.

The sight of each eye is defective, and glasses afford no relief. The pupils are equal in size, and respond fairly to the light. The conjunctivæ are very much injected. There is no arcus senilis. There is no squinting or ptosis.

The hearing is so dull as to make it necessary to speak in a very loud tone in order to be heard, and one ear seems to be as deaf as the other. There are also constant singing and humming noises in the head—"I still hear the wind in the shrouds," he says.

The memory is bad, the spirits are despondent, and of late (this statement is volunteered by the patient) there has been a frequent disposition to commit suicide.

The pulse is feeble, and about 70 in the minute. The appetite is good. The bowels are somewhat constipated, and a long time is spent over a stool. The urine is voided slowly, and with difficulty, although there is no stricture, and now and then it escapes during sleep. Sexually, the state may be spoken of as approaching to spermatorrhœa.

(b) Five years ago J. C—— began to suffer from pains in the legs and back, and to be unsteady in his gait; about the same time, also, his sight and hearing began to fail; and from that time to this he has continued to get gradually worse and worse. Four years ago he had sunstroke in the West Indies, of which the immediate symptoms were violent agitation and shaking, without loss of consciousness, and for which he was taken into a hospital and bled. But this accident was twelve months *after* his present malady had commenced, and therefore it is not possible to look upon it in the light of a cause. There never was either squinting or ptosis. He was at sea seventeen years in all, chiefly in hot climates, as the West Indies and the West Coast of Africa, and he remained on board three years after he had begun to suffer from unsteadiness of gait, and from the other symptoms which have been mentioned. Once during the time he was at sea he had chancres, without constitutional symptoms; and repeatedly he had diarrhœa; but, with these exceptions, his health on all occasions appears to have been pretty good. He says that he was always very careless, often sleeping, almost without clothes, on the bare deck, or on the ground, and that he was always "too much given to drink and women." For the last two years the sexual inclinations have been much damped, but before this time, from what he says, he appears to have been little better than a very satyr. Two years ago, when obliged to abandon his calling as a sailor, he was for a while treated in the hospital at Quebec for rheumatism. Afterwards he found his way to this country, and became an out-patient first at one hospital and then at another. During this time he appears to have been frequently blistered along the spine, and on one occasion to have been salivated. For the rest, I have only to add that his father died early in life of consumption; that his mother died young of some unknown chronic disease; and that a brother, the only child besides himself, is now dying of the disease which proved fatal to his father.

This case has not yet ended in a post-mortem examination; and of many other cases which have come under my notice, not one as yet is *complete* in this sense. All, therefore, that I can do is to say that in other cases of the kind the posterior columns of the spinal cord, and the posterior roots of the spinal nerves, are found to be diseased in the lumbo-dorsal region, and that the morbid appearances consist sometimes in a kind of grey degeneration, and sometimes in a gelatiniform and translucent condition, in a diminution of consistency, or in a state of induration called sclerosis. These changes are confined to the posterior columns of the cord; or if they extend further, it is not to the antero-lateral column, but only to the neighbouring portion of the posterior cornu of the central grey matter. In the majority of cases, the disease is confined to the lumbo-dorsal portion of the cord, and it is only in quite exceptional instances that it extends upwards, so as to implicate the cervical portion. In the majority of cases, the diseased structure is more vascular than the healthy structure of which it has taken the place, the vessels being more or less deeply imbedded in oil-globules of various sizes, and when examined further, it is found to be made up of atrophied and degenerated nerve-tissue, of the connective tissue in excess, and of amorphous granular matter. Now and then, also, traces of degeneration have been found at the roots or in the course of the optic nerves, or of one or other of the nerves of the muscles of the eye.

In order to see how far the case of which the notes have just been given agrees or disagrees with other cases of the kind, I single out, as points to be noticed in turn, the following:—Difficulty in standing or in moving about from incoordination of movement in the lower extremities; no true paralysis in the lower extremities; neuralgic pains, in the feet and legs more especially; more or less numbness in all forms of sensibility except that by which difference of temperature is recognised; impaired sight and hearing; no strabismus or ptosis; some incontinence of urine, and some want of control over the lower bowel, without marked paralysis of the bladder or sphincter ani; no obvious impairment of sexual power; no tingling or kindred phenomenon; no marked tremulous, convulsive, or spasmodic phenomena; no marked impairment of muscular nutrition and irritability; some impairment of mental and moral power; some injection of the conjunctivæ with contraction of the pupils; the sex and age; and, lastly, the frequent limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power, to the lower extremities.

Difficulty in standing or moving about from want of co-ordinating motor power to the lower extremities.—This difficulty is very evident, especially in the act of rising from a chair or in turning round suddenly when walking. If the patient cannot avail himself of some sufficient support at the time, the disorder in the movements of the legs produced by the act of rising from the sitting position, or of turning round suddenly when walking, is apt to throw him down. Walking is possible without a stick, but the gait is precipitate, staggering, the legs starting hither and thither vaguely, and the heels coming down at each step in a way which has gained for such patients at Gräfenburg the epithet of *stampers.* Moreover, it is less difficult to move on than to remain long in one position standing. In order either to stand or walk, however, the help of the sight is necessary. In less advanced forms of the disease, it may be difficult at first to detect incoordination in the movement of the legs, but this difficulty is not likely to last long. Often a first sign is reeling about upon getting out of bed in the dark. The patient may fall more than once under these circumstances, and think that he is only half awake or half sober. In that early stage of locomotor ataxy in which there is no evident incoordination of movement while the eyes are open, there is likely to be such disorder when the eyes are shut; and in an earlier stage still, even when it may be possible to stand steadily with the eyes shut, provided the patient be allowed to plant his feet where he pleases, it is more than likely that he will lose his balance if he be made to stand with the inner edges of the feet in close apposition. In more advanced stages of the disease, walking, or even standing, becomes altogether impossible, and it is curious to notice the extreme disorder in the movements of the legs when the patient is propped up under the arms, and made to try to walk or stand; for under these circumstances the legs are seen to go every way but the right way—backwards, forwards, sideways, unless it happens, as it often does, that they get foul of each other, and become interlocked. In all cases indeed, the incoordination of movement in the lower extremities, by which standing and moving about are interfered with in a greater or less degree, is a constant symptom in locomotor ataxy; and in a case of average severity, like the one under consideration, the gait, arising from this want of co-ordination, is quite characteristic—namely, precipitate, staggering, the legs starting hither and thither, and the heels coming down with a stamp at each step.

No true paralysis in the lower extremities.—When the patient is sitting or lying, he can, provided he sees what he is doing, move either leg singly, into any position with tolerable precision, and keep it there steadily; and the muscular force at his command is such, that it is out of my power to straighten the limb if bent, or to bend it if straightened. There is plainly no paralysis. Nor is it otherwise in other cases of locomotor ataxy, not even in those extreme cases in which the incoordination of movement in the legs has proceeded to the extent of making even standing an impossibility. And, certainly, it is no objection to the conclusion that my patient was speedily fatigued by the acts of standing or walking, for in reality this fatigue may easily be accounted for by referring it to the effort necessary to keep the ataxic movements of the legs in check.

Paroxysms of neuralgic pain, in the feet and legs principally.—My patient's chief complaint was of neuralgic pains—pains boring, stabbing, or shooting in their character, pains like those caused by a sharp electric shock—in various parts of the lower extremities, in the feet especially, and sometimes in the arms and abdomen, occurring in paroxysms varying in duration from a few minutes to many hours, flitting from one spot to another, but generally remaining at the same spot in the same paroxysm. And this was the chief complaint from the very beginning of the malady. Nor is this case at all exceptional in this respect: on the contrary, pain of the same character is met with in the great majority of cases of locomotor ataxy. Moreover, Dr. Trousseau speaks of this symptom as the most constant precursory phenomenon of the disease. In some cases, no doubt, pain is either absent altogether, or present only as an occasional symptom of very secondary importance. I have myself met with four cases of well-marked locomotor ataxy in which there was no pain, or none to speak of. The pain *may* begin in a way in which it may be mistaken for rheumatism, and be slow in acquiring its special character, but it has, as a rule, these special neuralgic characters from the first throughout.

Numbness in all the forms of sensibility excepting that by which differences of temperature are recognised.—In the case under consideration the sense of touch is almost annihilated in the soles of the feet and in the lower parts of the calves of both legs, and it is impaired greatly in the back of the thighs, in the nates to a less degree, and in the palms of both hands. In the parts also which are thus benumbed tickling is felt very obscurely, or not at all, and very trifling pain or none at all is caused by pinching or pricking. In the legs also the "muscular sense," as well as the special sensibility of the joints and bones, are considerably impaired, as is evident in the fact that the patient does not know where his feet are unless he can see them. Indeed, the only form of sensibility which seems to be unimpaired, is that by which differences of temperature are recognised. In other cases of locomotor ataxy, also, a similar state of things as to sensation would seem to be the almost constant rule, the numbness beginning, first in tactility, then in the sensibility to pain and tickling, afterwards passing to the "muscular sense," and always, curiously, skipping over, or leaving off before reaching, the sense by which differences of temperature are perceived. In some instances the sensibility of the mucous membrane of the anus and urethra is greatly deadened. The numbness is most marked in the lower extremities, especially in the feet, and very often it is confined to these parts, but now and then it may extend further. I know of one case in which the tip of the nose and the middle of the upper and lower lip are thus affected.

It would seem to be the rule for numbness to make its appearance at the same time as incoordination of movement, and for the two symptoms to make progress *pari passu*; but there are cases of locomotor ataxy in which, to say the least, numbness in any form is a very inconspicuous phenomenon. Moreover, it is certain that cases of well-marked locomotor ataxy are met with in which the "muscular sense" is not affected. Out of nineteen cases, I have met with two such. I believe, also, that in cases of locomotor ataxy in which the "muscular sense" is affected considerably, it will be often found that this form of numbness makes its appearance *after* the incoordination of movement, and not before it. In a word, I believe that the history of locomotor ataxy furnishes little countenance to a theory which has been advanced—that the incoordination of movement in this disorder is nothing more than the consequence of loss of muscular sense.

In some exceptional cases of locomotor ataxy there may be numbness in some parts, and an opposite state of things in others. Thus, I have myself met with a case in which there is anæsthesia almost complete in the lower extremities generally, and the most distressing hyperæsthesia as to tickling in the thumb. But, as I have said, cases of this kind are quite exceptional.

Impairment of sight and hearing.—Impairment of sight appears to be a common symptom in locomotor ataxy; impairment of hearing an occasional symptom. In the former case, Dr. Hughlings Jackson has shown that in the cases where sight is impaired or lost there is a *gradual* whitening of the optic disc without any marked change in the size of the retinal arteries and veins—a chronic form of atrophy which is more common in men than women, and which is not at all peculiar to locomotor ataxy.

Strabismus and ptosis.—Dr. Duchenne and Dr. Trousseau both speak of strabismus or ptosis as frequently met with in the early stage of locomotor ataxy, as frequently passing off after a time, and not unfrequently as returning, to remain permanently, at a later period. Dr. Duchenne has also twice met with paralysis of the fifth cranial nerve concurrently with paralysis of the third. Speaking of these symptoms, Dr. Trousseau says, "Some may be absent, but it rarely occurs that they are all absent in the same case. I have nearly always found them, and Dr. Duchenne is right in attaching great importance to them for diagnosing the disease at the onset. Remember, besides, that they may have been transitory, and been forgotten by the patient, so that the physician must needs make careful inquiries in order to discover their existence in the patient's previous history." Ptosis or strabismus was not present in the case which I have given, and never had been; and the same may be said of seven out of eighteen other cases of locomotor ataxy which have come under my notice. In the remaining eleven cases, strabismus or ptosis, one or both, were either present at the time of observation, or had been present for a time at an earlier period, generally at the onset of the disease. I find, also, as Dr. Trousseau did, that these paralytic affections of the muscles of the eye, or the impairment of sight or hearing, may be present at an early stage of the disease, may disappear for a while, and then reappear at a later stage.

No very obvious paralytic condition of the bladder or lower bowel.—Incontinence of urine at night, and now and then at other times, as after unusual fatigue, is a common and often a very early symptom in locomotor ataxy, and a less common, and usually a comparatively late symptom, is some trifling want of control over the lower bowel. Dr. Trousseau, speaking of the phenomena of the fully developed disease, says, "Just as in confirmed cases of paraplegia, there is paresis of the bladder and rectum, or even paralysis of the sphincters." As it seems to me, however, there is a marked difference between cases of confirmed locomotor ataxy and common paraplegia in these respects, the difference being that in locomotor ataxy there is not that obvious state of paralysis of the bladder, or sphincter ani, which is so generally present in paraplegia. Indeed, I have never met with a case of locomotor ataxy in which the way in which the bladder could be emptied in a steady stream, did not prove that this viscus retained a fair amount of power; and in one or two cases of this disease, in which the fæces have passed involuntarily at times, I have found a state of things which enabled me to account for this accident without assuming the existence of paralysis of the sphincter ani, namely, a want of sufficient sensitiveness about the anus. Moreover, I do not find in the cases which have come under my notice one in which the urine was retained, as it so often is in paraplegia, and where the consequences of such retention—cystitis, alkaline urine, and the rest—were present. Indeed, in all my cases the urine has been acid, and otherwise healthy—a state of things which is scarcely compatible with the presence of paralysis of the bladder.

No obvious impairment of sexual power.—From a sexual point of view, it is easy to see that, as a rule, there is a marked difference between locomotor ataxy and common paraplegia, the difference being that in the former disorder there is *not* that impairment of desire and power which is so constantly met with in the latter. Not unfrequently, indeed, it is plain that there is no impairment of sexual power in ataxy; and now and then there is a curious exaggeration of virility, evidenced, it may be, in the aptitude to repeated acts of connexion within a short period. Thus, Dr. Trousseau instances two cases in which these acts could be repeated as often as eight, nine, or ten times in a single night, and I have met with one case which is a fit fellow to these. In all these cases spermatorrhœa was a symptom. I also know of two cases of advanced locomotor ataxy in which fertilization has been successfully effected, and other cases of the kind are on record.

No tingling or kindred phenomena.—Tingling, or sensations analogous to tingling, are not among the symptoms noted in the cases of locomotor ataxy which have come under my own notice, and, so far as I know, they have not occurred in other cases of the kind. At any rate, I think it cannot be doubted that such symptoms are infinitely more common in common chronic paraplegia than in locomotor ataxy.

No obvious tremulous, convulsive, or spasmodic phenomena.—Dr. Trousseau says: "At an advanced period of locomotor ataxy, spasmodic contractions are frequently observed, not only when the patient wills a regular movement, but even in the state of rest. In the latter case, they consist in very powerful jerks of the limbs, and are an important symptom of this singular neurosis." But my experience of the disease does not bear out this statement. Moreover, the cases given by Dr. Bazire, in the valuable appendix to his translation of Dr. Trousseau's lecture on locomotor ataxy, is not confirmatory of the passage in the lecture which I have just quoted. Indeed, if I except certain attacks of convulsive agitation, in which one or two patients have now and then awakened out of sleep, and the feeling of constriction in the abdomen and lower extremities, which is occasionally met with, and which may possibly have some remote connexion with spasm, I know of nothing in the history of locomotor ataxy which requires a place in the category of tremulous, convulsive, or spasmodic phenomena.

No marked impairment of muscular nutrition and irritability.—This is another feature of locomotor ataxy, and, therefore, another point of difference between this affection and common paraplegia. The electro-sensibility is impaired in the muscles in which the "muscular sense" is impaired, not the electro-contractility.

Some impairment of mental and moral power.—Bad memory, despondency, suicidal tendency, are mentioned among the symptoms in the case which serves as my text, but troubles of this kind do not figure in the history of other cases of locomotor ataxy. In fact, it would seem to be the almost constant rule for the mental faculties to be unscathed in this disease.

Some injection of conjunctivæ with contraction of pupils.—In the case under consideration, the pupils were contracted and comparatively disobedient to light, and the whites of the eyes were considerably bloodshot; and this appears to be a not unusual state of things in cases of the kind. Dr. Trousseau says that he has often noticed in ataxic patients, in the intervals between the paroxysms of pain, injection of the conjunctivæ, sometimes as marked in the most violent conjunctivitis, sometimes amounting to a sort of chemosis, and, in association with this, a state of extreme contraction of the pupils; and he also tells us that he has seen this injection of the conjunctivæ and contraction of the pupils disappear during a paroxysm of pain. In J. C——, I failed to perceive this change during pain. Dr. Bazire also failed to perceive it in others who have come under his notice. I have observed it in two cases, of which that of my friend M. Ernst, the prince of violinists, was one. In these two cases, what I noticed was this—that the eyes ceased to be bloodshot, and the pupils opened when the pain reached a certain degree of severity and continued for a certain time, and not otherwise. This I observed on several occasions in M. Ernst while he was staying with me on a visit; and I expect that the discrepancy which at present exists between the statements of Dr. Trousseau and his translator upon this point, will disappear as soon as the influence of the degree and duration of the pain is taken into account.

The sex and age.—Locomotor ataxy is, without doubt, more common in males than in females. As regards sex, indeed, it is with this as it is with other disorders of the spinal cord; for out of 177 cases of all forms of disease of the spinal cord tabulated by Dr. Brown-Séquard, as Dr. Bazire pointed out, 128 occurred in men, and only 49 in women. Locomotor ataxy is also a disorder of adult life. In the cases which have come under my own notice the age ranges from 23 to 60, and but few cases are on record in which the patient was under 20. Indeed, the only cases under 20 would seem to be three reported by Dr. Friedrich of Heidelberg; of which the ages are respectively 18, 16, and 15 years.

The probable limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power) to the lower extremities.—In many cases of locomotor ataxy the upper extremities are not affected at all; in others, their sensibility is blunted in one form or other, and their movements are wanting in precision, especially if the sight be defective, or the eyelids closed. In the cases in which the movements of the upper extremities are wanting in precision, there is always, so far as I know, more or less impairment of sensibility, of the "muscular sense" perhaps most frequently; and my belief is, that the want of precision in movement is rather to be ascribed to the want of the proper guidance of sensation than to the loss of any co-ordinating motor power. One ground for this belief is the fact that the disease of the posterior columns of the cord which is met with in locomotor ataxy, and upon which, there is every reason to believe, the want of proper co-ordination in movement is dependent, is confined to the lumbo-dorsal region of the cord in the great mass of cases. Moreover, it is to be remembered that the movements of the arms in a biped-like man are not so interdependent as the movements of the legs, and that, on this account, movements of inco-ordination are less likely to occur in the arms than in the legs. It is also very possible that some of the cases in which the irregular movements of locomotor ataxy would seem to have extended from the legs to the arms may not have been true cases of locomotor ataxy. I remember one case of what at first seemed extreme locomotor ataxy, in which the arms were as much affected as the legs, but the patient in this case was totally blind and bedridden, and all but totally deprived of all kinds of sensibility in the arms, and of the "muscular sense" in the legs; and there was no difficulty in believing that the irregular movements of the arms (and possibly those of the legs also) were due, not to impairment in co-ordinating power, but simply to the muscular anæsthesia[1] and the blindness: and I do not remember any case in which the arms were affected in which the patient was not more or less in the same plight, as to muscular anæsthesia, if not as to blindness also.

Looking back, then, at the case which has been cited, and at the comments to which it has given rise, it is not difficult to see that locomotor ataxy is characterised by these symptoms :—

A peculiar gait arising from want of co-ordinating motor power in the lower extremities—a gait precipitate and staggering, the legs starting hither and thither in a very disorderly manner, and the heels coming down with a stamp at each step.

No true paralysis in the lower extremities or elsewhere.

Characteristic neuralgic pains, erratic, paroxysmal, in the feet and legs chiefly—pains of a boring, throbbing, shooting character, like those caused by a sharp electric shock.

More or less numbness, in the feet and legs chiefly, in all forms of sensibility, excepting that by which differences of temperature are recognised.

Frequent impairment of sight or hearing, one or both.

Frequent transitory or permanent strabismus or ptosis, one or both.

No very obvious paralysis of the bladder or lower bowel.

No necessary impairment of sexual power.

No tingling or kindred phenomenon.

No marked tremulous, convulsive, or spasmodic phenomena.

No marked impairment of muscular nutrition and irritability.

No impairment of the mental faculties.

Occasional injection of the conjunctivæ with contraction of the pupils.

The probable limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power) to the lower extremities.

For the rest, I will only say that a chronic disease with these characteristics, and without fever or other signs of disordered health, may safely be pronounced to be locomotor ataxy.

Dr. Duchenne, whose description of the disease is the best as well as the first, marks out three stages in locomotor ataxy. In the first stage the patient suffers from paralysis, often only temporary, of one or other of the motor nerves of the eye, from some degree of amaurosis, usually accompanied with unequal pupils, and from the peculiar neuralgic pains. In the second stage the characteristic unsteadiness in standing and moving about begins to show itself together with anæsthesia, the interval between the first stage and the second varying from a few months to several years. In the third stage the malady becomes more profound and general, but the precise point at which the second stage ends and the third stage begins is not very clearly defined. Dr. Duchenne does not regard the affection of the bladder, the rectum, and the genital apparatus as essential symptoms of the disease in any of these three stages; he regards them as *épiphénomènes* only. Dr. Trousseau does not divide the disease into distinct stages, but he speaks of a *premonitory* stage in which paroxysms of pain, spermatorrhœa or impotence, paralysis of one or other of the motor nerves of the eye, and disorder of vision are the symptoms to be met with. As Dr. Bazire says, however, "it is hardly possible to regard these merely in the light of premonitory symptoms, because they form part and parcel of the fully developed disease:" and, in fact, the various symptoms are so mixed up together, and make their *début* at such varying periods, that it is not easy to separate symptoms and arrange them in this stage or that.

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[1] *Vide* article on Muscular Anæsthesia.

2. CAUSES.—In some cases sexual excess would seem to figure as a cause, but not in others—not perhaps by any means in the majority. And this is one reason why it is not well to continue to use the name of *tabes dorsalis* as the equivalent of locomotor ataxy, for rightly or wrongly it has come to this, that the name *tabes dorsalis* is supposed to imply past abuse of the sexual organs. Nor is it possible to speak of syphilis, or rheumatism, or gout, or struma as a cause, for in a great many cases, to say the least, there is no evidence of one or other of these morbid conditions. In fact, it is not possible to refer locomotor ataxy to any special cause. What predisposes to other diseases of the nervous system predisposes to this, family predisposition especially, and this is all that can be said. With regard to family predisposition some curious instances might be given. I know of one case in which one brother is epileptic, another brother hypochondriac, and two sisters are suffering from different forms of paralysis; and Dr. Marius Carré instances a family in which eighteen members have become ataxic in turn,—namely, the grandmother, the mother, eight relations of the latter, seven children, and one cousin.

3. PROGNOSIS.—The prognosis of the disease is unhappily full of gloom. Usually, without doubt, the course is slowly but steadily in a downward direction—so slowly, often, that it is only after the lapse of many months, or even years, that the patient distinctly realizes the fact of having become decidedly worse; but on the other hand, several cases are on record in which the disease has advanced to the extent of destroying the power of standing and walking in four or five months.

Long pauses in the progress of the disease are not uncommon; thus, for example, I know of one case in which the condition has remained stationary for fourteen years. Moreover, it is not impossible to find a few cases in which the symptoms have changed for the better considerably, and are still changing. Cases of this kind, it is true, are not very common, but they are to be met with. I myself can testify to the existence of several of them.

4. Diagnosis.—Locomotor ataxy, it is said, may be confounded with several diseases, especially with common chronic paraplegia, with simple loss of "muscular sense," with cerebellar disease, and with chorea, but this can scarcely be if only moderate care be taken.

In common chronic paraplegia there is unequivocal paralysis in the lower extremities, and the nutrition and irritability of the paralysed muscles are, as a rule, unmistakeably impaired. In these fundamental particulars, indeed, the difference between this affection and locomotor ataxy is as complete as it can be. In common chronic paraplegia the bladder and sphincter ani are implicated in the paralysis which affects the legs, and the sexual power is almost sure to be greatly weakened or entirely extinguished. In common chronic paraplegia the characteristic neuralgic pains of locomotor ataxy are wanting, and numbness is nothing like so prominent a symptom as in the ataxic disorder. In common chronic paraplegia, where walking is possible, the gait, instead of exhibiting the want of co-ordination which is met with in locomotor ataxy, is hampered and slow, each leg being brought forward with evident difficulty even with the help of an upward hitch of the body on the same side, and the part of the foot first coming in contact with the ground being, as a rule, not the heel as in ataxy, but the toes. In common chronic paraplegia impairment of sight or hearing, strabismus or ptosis, injection of the conjunctivæ, or contraction of the pupils, frequent, if not constant, symptoms in ataxy, form no part of the history. In fact, in these respects, and in others of minor importance which might be mentioned, the histories of common chronic paraplegia and of locomotor ataxy are so different that it is not easy to see how, with only an ordinary amount of care, the two disorders can be confounded.

The ataxic movements which depend upon anæsthesia muscularis are only present when the patient does not see what he is doing: the ataxic movements which characterise *simple* locomotor ataxy continue whether the patient see what he is doing or not. Nor is this simple rule in diagnosis invalidated by the fact that in the majority of cases of locomotor ataxy the sight has a marked influence in keeping the unruly muscles in check, for the cases are almost exceptional in which loss of muscular sense does not form an important element in the disorder.

In some diseases of the cerebellum there appears to be, often at least, the same disorder of muscular movement which is met with in locomotor ataxy, but this resemblance is more apparent than real. In the next bed to that then occupied by the patient whose case has served as an instance of locomotor ataxy, was a boy, also under my care, whose cerebellum never seemed to have been properly developed, and whose gait was precisely that which I have seen in two cases of tumour of the cerebellum, and which seems to be associated with serious cerebellar disease in all cases. This boy reeled and rolled about in walking, but there was nothing peculiar in the way in which he moved his legs and planted his feet; on the contrary, these movements were those which would be instinctively made to prevent falling. He was not giddy, but merely unsteady, and the volitional and automatic movements of his legs were what they ought to be under the circumstances, no more. His mode of progression was widely different from that of the ataxic patient, as was at once apparent when the two were set to walk side by side; how different I need not again stay to say. In certain diseases of the cerebellum, also, some symptoms are likely to be present which will assist in the formation of a correct diagnosis, especially violent pain, often augmented by movement, in one or other part of the head, and frequent and obstinate vomiting, and at the same time other symptoms are likely to be absent which are present in locomotor ataxy; namely, neuralgic pains and anæsthesia in the feet and legs, and elsewhere.

In chorea there is great want of co-ordinating motor power, but the history is quite different from that of locomotor ataxy. Chorea is an affection of childhood and early youth; locomotor ataxy of adult life. The choreic muscular disturbances affect especially the head and arms; the ataxic are chiefly confined to the legs. Moreover, there are not in locomotor ataxy those involuntary movements which in chorea keep the affected muscles in a state of almost perpetual unrest. And as to the other symptoms, it is, in fact, a question of differences, not of resemblances.

It must not be forgotten, however, that the different diseases of the nervous system, like all other diseases, are not fenced in by any boundaries except those which have been fixed almost arbitrarily for the convenience of description, and that cases of a mixed character are continually being met with, which in reality lie across these boundaries in every direction.

5. TREATMENT.—The treatment of locomotor ataxy is not a subject upon which much can be said at present. No specific treatment can be recommended on good grounds, not even that by nitrate of silver, about which so much has been said of late in Germany and France; and the only treatment which finds favour in my eyes is one of a general character in which figure some preparation of phosphorus with or without cod-liver oil, or arsenic, or bichloride of mercury. I should endeavour to act upon general principles, meeting as well as I could any special indication, as syphilis, or gout, or rheumatism, or struma. I should trust to a liberal allowance of stimulants rather than to sedatives for the relief of pain; and for the relief of pain also I should

have much confidence in regular shampooing, in faradization, and the use of positive statical electricity. I am also disposed to think that good may be done by the use of irons or crutches, one or both. What is chiefly at fault is the motor power by which the two legs act in concert in standing and moving about: and what is wanted primarily is to do away, as far as possible, with the necessity for calling into exercise this power until it can have had time to recover by rest. This is an intelligible indication, and the use of irons or crutches is an intelligible means of carrying it out. Perhaps it is too much to expect that great good can be done in any way in advanced stages of the disease: but in early stages I cannot but think that the disease, to say the least, might be arrested if the patient would consent for a longer or shorter time to the use of these means. For surely it must go far to neutralize the good to be derived from treatment if the patient is continually trying to do, by walking about without help, or with only the imperfect support of a stick, what the diseased condition of his spinal cord incapacitates him from doing. Nor are these remarks alone applicable to the treatment of locomotor ataxy; on the contrary, they apply equally to the treatment of all forms of spinal disease in which the acts of standing and moving about are at all compromised.

VI. Spinal Irritation.

The first important work on the disorder now generally known as *spinal irritation* was published by Mr. Teale, of Leeds, more than forty years ago;[1] the next by the brothers Dr. and Mr. Griffin, of Limerick, about fifteen years later.[2] To Mr. Teale, indeed, belongs the credit of being the first to direct attention to this disorder, for, in reality, his claim either to priority or originality is scarcely, if at all, invalidated by the short communications which were made previously to medical periodical literature by Mr. Player, of Malmsbury,[3] by Dr. Brown, of Glasgow,[4] by Dr. Darwell, of Birmingham,[5] and still less so by anything written about the commencement of the century by Franks, Nicod, Ludwig, and others. It would also seem to be difficult to find any work of more recent date which deserves to be mentioned as at all equal in merit and importance to that of the brothers Griffin. The name "spinal irritation" was first proposed by Dr. Brown, of Glasgow.

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[1] A Treatise on Neuralgic Diseases dependent upon Irritation of the Spinal Marrow and Ganglia of the Sympathetic Nerve. By T. P. Teale. 8vo. London: Highley, 1829.

[2] Observations on the Functional Affections of the Spinal Cord and Ganglionic System of Nerves, in which their Identity with sympathetic, nervous, and irritative Diseases is illustrated. By William Griffin, M.D., and David Griffin. 8vo. London: Burgess and Hill. 1844.

[3] Quarterly Journal of Science, January 1822.

[4] Glasgow Medical Journal, May 1828.

[5] Midland Medical and Surgical Reporter, May 1829.

1. SYMPTOMS.—The symptoms of spinal irritation at first sight appear to be as vague and various as those of hysteria. They are in reality so far hysterical as to be not readily distinguishable. When further examined, however, one symptom stands out prominently, with which the others are obviously connected in a peculiar manner, namely, spinal tenderness; and the upshot of the whole matter appears to be that spinal irritation is a definite malady which must not be confounded with hysteria or with any other disorder. For example :—

Case.—In the early part of 1863, an unmarried lady, aged twenty-three, consulted me for pains in the head and face, loss of appetite, nausea, flatulence, palpitation, breathlessness, "sinking feelings," weakness, and low spirits. The pain, which was the chief suffering complained of, was sharp and neuralgic in its character, and varying in its seat, being sometimes in one part of the head or face, sometimes in another, and generally on the left side only. In the head it was often limited to a spot which might be covered with the tip of the finger, as in true clavus hystericus. Headache in one form or another was brought on or exaggerated by any effort, physical or mental: it was usually relieved by lying down and keeping perfectly still; it was scarcely ever absent except when face-ache had its turn; and sometimes it was so continuous and oppressive as to necessitate remaining in bed for days together. Nausea and sickness were its frequent accompaniment, and vomiting and great prostration were its common termination. In the cervical region of the spine there were considerable tenderness and a disagreeable feeling of weight, and pressure there brought on or increased the headache—the pain shooting from the occiput forwards—and caused a feeling of great nausea and oppression at the præcordia. The feet were always cold; "chills and flushes" were of frequent occurrence, and so were yawning, sighing, and stretching of the arms. Sleep was often made hideous by nightmare; fits of lowness of spirits and crying, attended by a sense of choking, as from a ball or knot in the throat, and followed by plentiful gushes of pale, limpid urine, were brought on by the most trivial causes; and the manner and appearance were altogether those of an eminently nervous or hysterical person. Menstruation was regular, neither excessive nor deficient, and it could not be said that the sufferings were either more or less at this time. The bowels also acted properly, and (but for the disposition to pass large quantities of pale urine, which has been already mentioned) so did the kidneys and bladder.

These symptoms, it appears, had their starting-point about twelve years ago in the shock and grief caused by witnessing the death of a brother, her last remaining near relative, in an epileptic fit, and ever since this time they have continued very much as they now are, with but little intermission. Before this time the personal history of the patient was tolerably good, but not so her family history; for, in addition to the brother whose death in epilepsy has just been mentioned, it appears that her father died years before of phthisis, and that her mother is now in a lunatic asylum.

Under the use of a more liberal diet, with ammonia and calumba, and with occasional blisters to the nape of the neck, health was re-established in little more than a month, notwithstanding the fact that several days at the commencement were wasted in overcoming a dislike to take the wine and medicine necessary—in converting, in fact, the patient from a firm belief in teetotalism and homœopathy.

Towards the close of the same year, 1863, this young lady again returned to me, looking very worn and thin, with all her old symptoms in force, and with cough and difficulty of breathing in addition. The cough was very violent, barking, unattended with expectoration, and often carried on until it ended in retching and vomiting. The difficulty of breathing was chiefly at night: usually it did not amount to more than what might be met by a voluntary effort at inspiration; now and then it seemed to deserve the name of asthma; almost invariably it was accompanied, not by a feeling of a ball or knot in the throat, but by a sharp pain in the left hypochondrium, or else by severe aching in the left shoulder and down the left arm. Percussion and auscultation failed to bring to light any signs of disease in the heart or lungs, but pressure along the spine revealed tenderness in the cervical and upper dorsal regions, in the latter especially, and at the same time brought on cough, deep inspirations, pain and throbbing at the epigastrium, and a feeling of great faintness and breathlessness.

On this occasion a very fair state of health was soon re-established by the plan of treatment which proved successful in the first instance.

At the beginning of 1865, this lady again required my services. For the three weeks before my seeing her she had been in bed, with her thighs drawn up tightly against her abdomen, and with her heels buried in her nates. This contraction was unremitting during the waking state, and only partially remitting during sleep: it was unattended by pain; and it could be partially overcome, for a time, without causing much pain in the contracted muscles, by slow and steady extension. The headache and face-ache had gone months before, and so had the pain in the epigastrium and in the left shoulder and arm; the cough and difficulty of breathing and palpitation were of very unfrequent occurrence; the appetite and digestion and the action of the bowels were tolerably natural; and what was complained of now were colicky pains in the lower part of the abdomen, pains often very severe and sickening about the loins and hips, and in the region of the left ovary, with constant calls to pass water, and much pain in the urethra in attending to these calls. The spine was now tender, not as before in the cervical and dorsal region but low down in the lumbar region, and pressure on the tender part brought on colicky pains in the lower part of the abdomen, and a cutting pain in the urethra, with an almost irresistible impulse to pass water then and there. Pressure in the cervical and dorsal regions of the spine gave rise, not to the marked symptoms produced in this way in the two previous illnesses, but simply to a disagreeable thrill all over the body. There was no numbness or tingling in the legs or elsewhere, and no hyperæsthesia, except perhaps to a very trifling degree over the left ovary. Tickling the soles of the feet gave rise to painful spasmodic shocks in the legs, to a disagreeable thrill passing up the body as high as the throat, and to the involuntary escape of a small quantity of urine. The condition as to general health was tolerably good—much better than during the two previous illnesses: and, in fact, the only sign of disorder, in addition to those which have been indicated (and this can scarcely be reckoned as such), was the absence of menstruation since the birth of a child about three months ago.

Somewhat more than twelve months ago, after having been quite well for the year previously, this patient married and became pregnant. In the early months of pregnancy she had much headache, depression, weakness, and sickness; but after a while these symptoms passed off, and everything went on smoothly and satisfactorily until two months after confinement, when her baby died suddenly. And then began her present troubles. The fretting about her baby brought back the old headaches, the headaches produced great sleeplessness and irritability of the stomach, and then came on a state of uncontrollable fidgetiness which kept her incessantly moving about until her legs, one leg especially, failed altogether, and obliged her to take to her bed. The very next morning her legs had become contracted, and she herself is convinced that this change for the worse, as she regards it, was brought about by the pain and loss of blood produced by introducing a large speculum and by applying leeches to the os uteri on the previous evening.

The treatment on this occasion consisted chiefly in a liberal allowance of food and wine, in repeated blisterings to the lumbar region of the spine, and in the administration of bromide of potassium and ammonia; the result was the cessation of the contractions in about three weeks, and the complete re-establishment of health in about two months and a half.

In commenting upon this case with the view of separating the general phenomena of spinal irritation from the particular, I take the following as the points which most deserve to be attended to, namely these:—Spinal tenderness, neuralgia, spasmodic cough and difficulty of breathing, palpitation and vascular throbbings, nausea, vomiting and eructations, and irritability of the bladder, all in connexion with spinal tenderness; the connexion of particular symptoms or groups of symptoms with tenderness in particular parts of the spine; prolonged muscular contraction; no paralysis of the limbs; no paralysis of the bladder or rectum; no numbness; variability and inconstancy of the symptoms; a nervous constitution.

Spinal tenderness.—In the great majority of cases this symptom would seem to be present in spinal irritation and absent in spinal meningitis, myelitis, or spinal congestion, acute or chronic. It would seem, indeed, to deserve to be regarded as the pathognomic symptom of spinal irritation; for in the few cases of spinal meningitis, myelitis, or spinal congestion in which it is met with, there is reason to believe that its presence may be accounted for by the association of the phenomena of irritation with those of inflammation or congestion. At any rate, it is certainly the rule that spinal irritation without spinal inflammation or congestion is accompanied by spinal tenderness, and that spinal inflammation and congestion without spinal irritation is *not* accompanied by spinal tenderness. Spinal tenderness, however, can scarcely be spoken of as a prominent symptom in spinal irritation. It is often not complained of until it is specially inquired after; and now and then its existence is not even suspected by the patient until he or she is made to wince under pressure applied to the spine. In a few cases which from their symptoms would seem to come under no other head than that of spinal irritation, there is no spinal tenderness—only five such cases are met with among the 148 cases brought together by the brothers Griffin, and these may without difficulty be in great measure explained away; but such cases are much too exceptional and doubtful to throw discredit on the rule in question, that spinal irritation and spinal tenderness go together. Spinal tenderness, however, does not appear to be equally marked in all forms of spinal irritation. It appears to be much less marked where the irritation shows itself in spasm and prolonged muscular contraction than in the cases where it shows itself in pain; and it is certainly absent in tetanus, which in one sense may be looked upon as the manifestation of spinal irritation in its most aggravated form.

Nervous pains, often in connexion with tenderness in a particular part of the spine.—Nervous pains, neuralgias, in one place or another, often intermittent and more or less regularly periodical, and often shifting suddenly from one place to another, are a very common, perhaps the most common, symptom in spinal irritation. They are often brought on or exaggerated by lifting any weight, by twisting or straining the back in any way, or by any effort, mental or physical: and as often they are relieved, to some extent at least, by lying down. Very often, also, there is tenderness in the portion of the spine corresponding to the insertion of the affected nerves—in the upper cervical region, where the pains are in the scalp (clavus hystericus, megrim, and others), face, or neck; in the lower cervical region, where they are in the upper extremities, shoulders, and upper part of the thorax; in the dorsal region, where they are in the lower part of the thorax and upper part of the abdomen (pleurodynia, gastrodynia, infra-mammary stitch, and others); in the lumbar and cervical regions, where they are on the lower part of the abdomen, hips, loins, and lower extremities. In the majority of cases the pain would not seem to be in the part of the spine which is tender, or in any other part. In some cases there may be aching in some part of the spine, or else a sense of weight and heat; but I am very much inclined to believe that these last mentioned symptoms, and "back-ache" generally, have often to be referred to spinal congestion rather than to spinal irritation in its uncomplicated form. When the spinal tenderness is very great, slight pressure will often cause pain to strike from the tender spot of the spine to the distant seat of pain, or will bring about or exaggerate this pain. This fact is illustrated in the case I have given, and better still in some of the cases related by the Griffin brothers. In one of these cases, for example, where the whole spinal column was found to be acutely tender, "pressure of the first or second vertebra occasioned pain, which shot forwards from the occiput to the brow; a little lower, pain was excited at the larynx; on pressing one of the lower cervical, it occurred at the point where it dips behind the sternum; on pressing the upper dorsal, at the middle of the sternum; from the third or fourth dorsal to the eight or ninth, it was excited at the ensiform cartilage; yet lower, at the sides; and in the lumbar vertebræ, pain was excited in the iliac and pubic regions" (p. 19). And in another case, where there was some tenderness of the middle cervical vertebræ, and acute tenderness from the fourth dorsal to the eighth or ninth, "pressure on any of those last, especially the seventh or eighth, brought on violent pain, which darted forwards to the ensiform cartilage. When the last-mentioned vertebra was pressed upon, the patient said that she thought her 'heart would break'" (p. 119). The pain is often curiously localised: sometimes it gives the idea of a nail being driven into the part, as in clavus hystericus; sometimes the feeling produced by it is as if a walnut or other hard substance were pressed under a tight belt; sometimes it is very severe, and neuralgic in its character rather than rheumatic: and not unfrequently, when it has existed some time, the painful part becomes tender on pressure. Most generally this morbid sensation is in the form of pain, but now and then it may take that of cold, tingling, itching, or some other feeling which is disagreeable rather than painful. The amount of constitutional disturbance attending the pain varies very much, but it is usually comparatively trifling, and, as it would seem, quite out of proportion to the degree of suffering.

Nausea, retching, vomiting, eructation, &c. often in connexion with tenderness in a particular part of the spine.—These are common symptoms in spinal irritation: next to pain, indeed, they are perhaps the most common. They are also intimately connected with certain forms of pain, especially cephalalgia and gastrodynia, sometimes preceding, sometimes accompanying, but more generally following, the pain. As regards the particular part of the spine which is likely to be tender when the stomach is the seat of irritation, the Griffin brothers say that "nausea and vomiting appear to bear more relation to tenderness of the cervical spine, pain of stomach to tenderness of the dorsal; but that where there was soreness of both, nausea and vomiting was still more frequent, and pain of stomach scarcely ever absent." The epigastric disorder in these cases is generally accompanied with tenderness on pressure, not merely in the spine but also in the epigastrium and in the left hypochondrium—with those three patches of tenderness which M. Briquet speaks of as the "trépied hystérique"—as the tripod upon which the diagnosis of hysteria rests.

Spasmodic cough, difficulty of breathing, &c. often in connexion with tenderness in a particular part of the spine.—These again are symptoms which are common enough in spinal irritation, and mostly so, as it would seem, when the tenderness in the spine is in the cervical and upper dorsal region.

Palpitation, &c. often in connexion with tenderness in a particular part of the spine.—Palpitation is another symptom of spinal irritation which seems to be oftenest met with when there is tenderness in the upper half of the spine. It seems to be not unfrequently associated with a feeling of epigastric pulsation, and with nausea, vomiting, and other signs of gastric disorder. Vascular throbbings in other places, as in the temples, and "chills and flushes," and a disposition to syncope, and other signs of disturbed balance in the circulation, may, and often do, go hand in hand with the palpitation, and seem to have to do with the same condition of the spine.

Irritability of the bladder, often in connexion with tenderness in a particular part of the spine.—This was a marked symptom in the case which I have related when the seat of spinal tenderness shifted to the lumbar region, and it seems to be a very common, if not a constant symptom, in cases in which the tenderness is in this region.

The connexion of particular symptoms or groups of symptoms with tenderness in particular regions of the spine.—The data best calculated to illustrate this connexion are those supplied by the brothers Griffin. These consist of no less than 148 cases, of which 26 are in males, 49 in married women, and 73 in girls. In these 148 cases, the spinal tenderness was in the cervical region in 28, in the cervical and upper dorsal region in 46, in the dorsal region in 23, in the dorsal and lumbar region in 15, in the lumbar region in 13, and in the spine generally in 23. In the following table the prominent symptoms connected with each one of these forms of spinal tenderness are set forth in a way which requires no comment except this—that this grouping of symptoms with tenderness in particular parts of the spine must only be looked upon as approximating to the truth, and that now and then any symptom may appear out of the order in which it is set down.

Region of Spinal Tenderness.	*Prominent Symptoms.*
A. *Cervical region.* Cases 28 in number.	Headache, nausea, vomiting, face-ache, fits of insensibility, cough, pains in the upper extremities, &c. ⁂ Nausea and vomiting in 5 cases, pains of stomach in 2 only.

B. *Cervical and Dorsal region.* Cases 46 in number.	In addition to the symptoms in group A, pain of stomach and sides, pyrosis, palpitation, oppression. ⁂ Pain of stomach in 34 cases, nausea and vomiting in 10.
C. *Dorsal region.* Cases 23 in number.	Pain in the stomach and sides, cough, oppression, fits of syncope, hiccup, eructations. ⁂ Pain in the stomach in almost all these cases, nausea or vomiting in only one.
D. *Dorsal and Lumbar region.* Cases 15 in number.	In addition to the symptoms in group C, pains in the abdomen, loins, hips, lower extremities, dysury, and ischury. ⁂ Nausea in only one case.
E. *Lumbar region.* Cases 13 in number.	Pains in the lower part of the abdomen, testes, or lower extremities, dysury, ischury, disposition to paralysis in lower extremities. ⁂ Retching and spasm of the stomach in one case only.
F. *All regions together.* Cases 23 in number.	A combination of the foregoing groups of symptoms, one group changing into another as the spinal tenderness becomes more marked in one region than in another.

Prolonged muscular contraction.—This is a very conspicuous symptom in the case which serves as my text, and it is no uncommon symptom in other cases of spinal irritation. The lower extremities appear to be the parts most commonly affected, one or both of them; but the upper extremities can claim no exception, nor even the muscles of the jaws and neck, trismus or torticollis being among the results in this latter case. "Occasionally," says Mr. Teale, "there is an inability to perform complete extension of the elbow, the arm appearing restrained by the tendon of the biceps, pain and tightness being produced in this part when extension is attempted beyond a certain point;" and to this fact I can testify. Moreover, I can testify as to the not unfrequent occurrence of long-continued closing of the fingers and thumb upon the palm. The rule appears to be, for the extremities to be affected before the trunk or head.

This contraction, which is generally painless, may be prolonged for weeks or even months continuously, even during sleep, or with occasional intermissions of uncertain duration; and the attacks, primary or secondary, are usually found to begin and end suddenly and unexpectedly. The relations between this form of contraction and that which occurs in other cases, especially in tetanus and in that somewhat vague disorder to which Dr. Trousseau has given the name of tetany (tétanie), are not very easily determined. In tetanus, with very rare exceptions, the contraction is painful, especially in the paroxysmal bouts, and the order in which it attacks the body is different —first, the jaws; then the trunk; and the extremities only at a late period, if at all. In tetany, as in tetanus, the contraction is painful, but the order in which the body is attacked is different to that which is observed in tetanus, centripetal not centrifugal,—first the extremities, then the trunk or head; the contraction, in fact, being confined to the extremities, except in cases of unusual severity. In the way in which it affects the extremities first, and often exclusively, the contraction of tetany agrees with the contraction under consideration, but in other respects it differs. It differs, especially, in being ushered in and accompanied by symptoms which do not seem to form part and parcel of simple spinal irritation; namely, tingling and some degree of anæsthesia, and also (so it is said) in the form of the contracted hand being peculiar—like that which the hand of the accoucheur takes in order to be introduced into the vagina—and in the possibility of bringing on the contraction by firm pressure upon the principal nerves or arteries of the affected muscles. It may be questioned, however, whether there are absolutely fixed lines of division between these different forms of prolonged contraction, and whether the difference which exists may not be accounted for as the result of different degrees of irritation, affecting, it may be, different parts of the spinal cord. It may be questioned, also, whether a sufficient case is made out for describing tetany as a definite disorder, and whether it is not rather a form of spinal irritation complicated with some graver spinal disease—spinal meningitis, myelitis, spinal congestion—in varying proportions. The association of tingling and numbness with the prolonged contraction is, as it seems to me, a reason for an affirmative conclusion. At any rate, prolonged muscular contraction, be its significancy in tetanus or tetany what it may, must be looked upon as a not unfrequent symptom in simple spinal irritation—as a symptom, too, which is usually of no very grave import. Of this there need be no doubt.

No paralysis of the limbs.—In the case I have given in illustration there was great weakness of the legs, and one leg seemed to "drag" immediately before the contractions came on. There was a disposition to paralysis in the legs, but not more than this; nor do I find paralysis of the limbs among the symptoms of spinal irritation strictly so called. There is, no doubt, a connexion between paralysis and spinal irritation which cannot be overlooked; and under that form of paralysis which is known as "hysterical paralysis," and about which more will have to be said in due time, and under spinal irritation, there is a common basis. As it seems to me, however, it is pathologically as well as physiologically incorrect to speak of hysterical paralysis as a symptom of spinal irritation. Also, it seems to me, the right place of this paralysis is after spinal irritation, not along with it, when the capability of morbid action which is implied in the term irritation is worn out; and so in the other exceptional cases in which paralysis is connected with spinal irritation, it will, I believe, be found on careful examination that the paralysis is not a symptom of actual spinal irritation, but of a state of vascular change into which this irritation may issue and has issued—spinal congestion, it may be, or even myelitis.

No paralysis of the bladder or bowel.—The remarks which have just been made apply equally to paralysis of the bladder or sphincter ani. Paralysis in either of these parts, or even a disposition to it, is rarely met with in any case which can be strictly brought under the head of spinal irritation; and in the few exceptional instances which do occur, it is plain enough, when the matter is fairly inquired into, that the boundary has been passed which separates the state of irritation from the state of exhaustion, and that, in fact, the case is no longer one of simple spinal irritation.

No numbness.—Numbness, again, is a symptom which is scarcely ever met with in cases to which the name of spinal irritation is strictly applicable, and, when it is met with, it is easily accounted for. In short, the relationship of numbness and paralysis to spinal irritation appears to be one and the same, the numbness and the paralysis being alike connected, not with the state of morbid action called irritation, but with the after-state of morbid inaction for which exhaustion seems to be one of the appropriate names.

Variability and inconstancy of symptoms.—One most characteristic feature of spinal irritation is the way in which one symptom or group of symptoms may change, and change suddenly, into another symptom or group of symptoms. It is now this disease which is simulated, now that, there being scarcely any disease which may not be copied: at one time the head is affected, at another the chest, at another the abdomen or the extremities: and the only thing constant among these ever-shifting phenomena appears to be this—that the spinal tenderness changes from one part to another in a manner which is intelligible enough when the connexion of the spinal nerves with the affected part is taken into consideration.

A nervous constitution.—The subjects of spinal irritation, with few if any exceptions, may be spoken of as hysterical, hypochondriacal, or nervous. They have, in fact, that nervous constitution which Whytt, following in the steps of Sydenham, showed to be the common basis of hysteria and hypochondriasis. First in order among the signs of this constitution comes that sign which Sydenham regarded as pathognomic of hysteria and hypochondriasis—namely, a proneness to pass, under or after strong emotion or excitement, large quantities of pale limpid urine. Then come other signs scarcely less characteristic: proneness to tenderness, not only in some part of the spinal column, but also in the epigastrium and left hypochondrium—*le trépied hystérique* of Dr. Briquet already referred to; proneness to sudden and distressing flatulent distension of the stomach and bowels, with loud rumblings and explosions, and with a feeling of a ball rolling about, first in the left flank, and then mounting, or tending to mount, into the throat, where it gives rise to a sense of choking and to repeated acts of swallowing; proneness to bursts of crying and sobbing or of laughing; proneness to sighing, yawning, and stretching the arms; and proneness to fits of convulsive agitation and struggling. Then comes a promiscuous series of signs: proneness to erratic pains of a neuralgic character, breathlessness, nervous cough, palpitation, throbbing in the temples, epigastrium, and elsewhere; "flushes and chills," syncope, hiccup, nausea, vomiting, aversion to food or unnatural craving for it, heartburn, oppression at the præcordia, languor, debility, fidgetiness, tremulousness, vertigo (especially on rising hastily), ringing in the ears, fickleness, fancifulness and inability to discriminate between fact and fiction, undue lowness of spirits or the contrary, and other symptoms whose name is legion. Not only, indeed, is the name of these different symptoms legion, but there is ever going on a process of mutual metamorphosis in the symptoms themselves; and, in conclusion, it is this very hysterical or hypochondriacal variability and mutability of the symptoms which must be looked upon as the great characteristic of the nervous constitution.

2. Post-mortem Appearances.—The morbid structural changes strictly belonging to spinal irritation are *nil*. The disease is nervous or functional in its character, and on this account it leaves no obvious traces after death. Still, as Dr. Copland wisely says, "an affection which may with justice be viewed as functional to-day—as spinal irritation merely—may be inflammation on the morrow, and rapidly followed by the consequences of inflammation." Such a termination, however, is altogether exceptional: and when it does occur, the history during life will show very clearly that any traces of inflammation which are met with after death are to be ascribed, not to irritation, but to inflammation. How far irritation, which involves in its very essence, as I believe, capillary contraction and bloodlessness, not capillary paralysis and congestion, may involve changes which are opposed to inflammation—deficiency of blood and organic changes brought on by the part being starved for want of blood—remains to be seen. I take it that such changes would have been found if they had been looked for with the same amount of care which has been expended in the search for inflammatory changes: but the investigations have yet to be made which will verify or disprove this conjecture.

3. CAUSES.—Neglect of gymnastic training, insufficiency of wine or other alcoholic drinks, over-indulgence in sexual matters, onanism, would seem to deserve a conspicuous place among the causes of spinal irritation. It is idle, however, to weigh the importance of particular causes, or even to attempt to individualize them, and it is enough to be content with the broad fact that everything which tends to induce a nervous habit,—that is, everything which exhausts vital power,—must be reckoned as a cause. I believe that the starting-point of the disorder will very often be found in some strain or blow to the back, and I also believe that a congenital predisposition may also be detected in very many cases.

4. DIAGNOSIS.—The fundamental question for consideration in this place is how to distinguish between functional and organic affections of the spinal cord, and this question fortunately is one which is less difficult to answer than it might seem to be at first sight. In fact, the characteristics of spinal irritation indicated by the Griffin brothers are sufficient of themselves to supply the answer to any one who has tolerably clear ideas respecting the principal diseases with which spinal irritation may be confounded. These characteristics are:—'1st. The pain or disorder of any particular organ being altogether out of proportion to the constitutional disturbance. 2d. The complaints, whatever they may be, being usually relieved by the recumbent posture, and always increased by lifting weights, bending, stooping, or twisting the spine. 3d. The existence of tenderness at that part of the spine which corresponds with the disordered organ, and the increase of pain in that organ by pressure on the corresponding region of the spine. 4th. The disposition to the sudden transference of the disordered action from one organ or part to another, or the occurrence of hysterical symptoms in affections apparently acute; and 5th. The occurrence of fits of yawning or sneezing, which, though not very common symptoms, yet, as rarely ever occurring in acute organic disease, may generally be considered as characteristics of nervous irritation."

In the diseases of the spinal cord which have already been under consideration—spinal meningitis, myelitis, spinal congestion, and tetanus—it has been seen that it is the rule for the spine *not* to be tender on pressure, and in spinal irritation it has been seen that such tenderness is so constant as to deserve being reckoned as the distinctive feature. Here, then, is a point of difference which will serve as a guide to a correct diagnosis in several important cases in which guidance is necessary,—which will serve as a guide in almost all cases except in that with which spinal irritation is most readily confounded. This case, which is strumous disease of the vertebræ, is one in which spinal tenderness is also present, as well as many other symptoms of spinal irritation,—pain in the side, stomach, or bowels, cough, oppression, tightness around the waist, and so on—and in which relief is obtained by reclining. Nay, there may even be in spinal irritation a yielding and projection of the tender vertebræ, with some puffiness of the overlying skin, which simulates in no imperfect manner the earlier stage of angular curvature. There are many resemblances, in fact, but, as the brothers Griffin have pointed out, there are also certain differences which are so well marked as not to leave the diagnosis in doubt. Thus it is found:—"1st. That strumous disease of the vertebræ attacks the young, and most frequently those under the age of puberty, who are least of all liable to be affected by spinal irritation. 2d. That disease of the vertebræ, when attacking young girls, is seldom accompanied by symptoms of a purely hysterical character, while any serious irritation of the cord can scarcely exist without them. 3d. That an apparent prominence of the tender portion of the spine, which sometimes exists in cases of irritation, is never strictly angular; for, if four or five of the vertebræ seem to project, the prominence is nearly equal in all, whereas in caries of the bones it is greatest in the middle, the prominence depending, in fact, on a slight puffing of the ligaments or investments of the spine, and not on displacement or curvature. 4th. That absolute paralysis of the lower limbs is a rare consequence of irritation, and a frequent one of caries of the bones. 5th. That the general health suffers less in the former complaint, and it is not attended with the look of serious organic disease which is indicative of the latter. 6th. That the constitution of the patient may also prove useful as a guide, the disposition to spinal irritation, as well as to scrofula, being hereditary."

5. Prognosis.—However urgent the symptoms may be, the prognosis in spinal irritation is favourable rather than unfavourable. It must always be borne in mind, however, that spinal irritation is a state which may issue in inflammatory or other organic changes in the cord or in its membranes, and that a favourable prognosis must be qualified by this contingency, especially in those cases in which there is some obvious vice of the constitution—scrofulous, gouty, rheumatic, syphilitic, or other.

6. Treatment.—"Local depletion by leeches or cupping," says Mr. Teale, "and counter-irritation by blisters to the affected portion of the spine, are the principal remedies. A great number of cases will frequently yield to the single application of any of these means. Some cases, which have even existed for several months, I have seen perfectly relieved by the single application of a blister to the spine, although the local pains have been ineffectually treated by a variety of remedies for a great length of time." Of the efficacy of blisters in these cases I have had abundant proof. As to the good effects of local depletion I have had less experience, partly because I found that the blisters were sufficient of themselves, and partly because I believe that the state of irritation is associated with a state of capillary contraction and bloodlessness, and not with a state of capillary

paralysis and congestion. Still, I can well believe that there are many mixed cases in which irritation has issued in some degree of capillary paralysis and congestion, especially in the skin at the seat of spinal tenderness, and in which this state will be greatly relieved by local depletion.

As regards medicine, I should certainly be disposed to trust most in common tonics—quinine, steel, or cod-liver oil; to the latter in conjunction with some preparation of phosphorus most of all, perhaps. And certainly I should be disposed to fight against pain and spasm, as I have sufficiently explained elsewhere, by remedies which rouse the circulation to greater activity, and not by those which have a contrary action. Nay, I should even have more confidence, as a local application for pain, in some application which would produce a hyperæmic condition of the skin, than in any one which had a deadening effect upon the sensitiveness of the part.

It is, no doubt, an indispensable part of the treatment to avoid standing or walking to the extent of producing fatigue, but there would seem to be no necessity, except as a very temporary measure perhaps, to insist upon a recumbent position being retained for any length of time. Upon this point Mr. Teale says (and he says all that need be said), "When my attention was first directed to this subject, I considered recumbency a necessary part of the treatment: it is, for a moderate length of time, undoubtedly beneficial, and frequently very much accelerates recovery; but subsequent observation has convinced me that it is by no means essential. I have seen several instances of the most severe forms of these complaints, occurring in the poorer classes of society, where continued recumbency was impracticable, which have, nevertheless, yielded without difficulty to the other means of the treatment, whilst the individuals were pursuing their laborious avocations."

As regards diet I have only this to say—that I believe the great thing to be done is to supply wine or some other alcoholic drink as well as nutritious food in sufficient quantity. I believe that nutritious food in itself is not enough. In very many cases it is found that alcoholic drinks are either abstained from altogether or taken in very insignificant quantities from a fear that they will aggravate the pain or spasm, or for some other) reason: in very many cases it is found also that relief is obtained only when this practice is abandoned, and the diet made to include at least an average share of the drinks in question. Indeed, the result of my own experience is unequivocal in this respect—that the somewhat bold use of alcoholic drinks is a cardinal point in the treatment of spinal irritation, and that this indication must be fully acted upon if this treatment is to lead to anything like satisfactory results.

Of the spinal maladies remaining to be noticed the principal are these:—General spinal paralysis, hysterical paraplegia, reflex paraplegia, infantile paralysis, hæmorrhage, non-inflammatory softening, induration, atrophy, hypertrophy, tumour, concussion, compression, caries of the vertebral column, spina bifida, &c.

VII. General Spinal Paralysis.

There is a form of general paralysis to which Dr. Calmeil gave the name of general paralysis of the insane, and with which all who know anything of insanity are sufficiently familiar. It may co-exist with any form of insanity, but it is most commonly associated with the monomania in which the patient believes himself to be possessed of unbounded opulence. The first signs are likely to be thickness of speech, quivering of the lips and tongue, fumbling and clumsy movements of the fingers, with an unsteady and sideling gait. Then the urine escapes now and then involuntarily, or even the fæces. Once begun, the downward course of the malady is headlong, and in a few months, in a few weeks it may be, within two or three years at the most, the patient is in bed, altogether without the power of supporting himself on his feet, unable to use his hands so as to help himself in any way, incapable of sitting up or even of turning over in bed, requiring to be fed like a child, and, when fed, in no small danger of choking if left to masticate the morsels, with urine and fæces escaping under him unheeded, and with every power of body and mind an utter wreck. With few exceptions the thickness of speech shows that the muscles of the tongue and lips are the first to fail, but in fact all parts of the muscular system show signs of weakness about the same time, and it is difficult to fix upon any one part and say that it is affected before the rest. Sometimes, the paralysed muscles become considerably atrophied, but the rule appears to be that such atrophy is less marked than in cases where the paralysis is the result of disease in the spinal cord: always, according to Dr. Duchenne, the paralysed muscles, whether atrophied or not, retain their full share of electro-contractility. After death signs of disease are found in the brain, but not in the spinal cord; these signs being increased vascularity, with serous or sero-fibrinous infiltration in the pia mater, in the cortical substance, and in the brain structure generally.

General spinal paralysis is the name used by Dr. Duchenne to describe a form of paralysis which, until he pointed out the differences, was confounded with general paralysis of the insane. Looking hastily at the phenomena of paralysis when clearly developed, it is, indeed, not to be wondered at that these two disorders should have been confounded; but in reality general spinal paralysis, as defined by Dr. Duchenne, possesses peculiarities which are sufficiently characteristic. In general spinal paralysis the mental faculties are natural; in general paralysis of the insane they are fundamentally deranged. In general spinal paralysis the electro-contractility of the paralysed muscles is abolished or greatly impaired; in general paralysis of the insane it is intact. In general spinal paralysis the paralysis usually begins in the legs and travels upwards, often remaining in the lower parts of the body a long time before attacking the tongue, face, and upper extremities; in general paralysis of the insane all parts of the muscular system would seem to be affected simultaneously, or, if there be any difference as to time, it is the tongue and the upper parts of the body which are the first to suffer. In general spinal paralysis there is a marked disposition to atrophy in the paralysed muscles and elsewhere, to bed-sores, and to other signs of defective nutrition; in general paralysis of the insane these evidences of wasting are, to say the least, far less conspicuous. In general spinal paralysis the progress of the disease is slow, often extending over several years; in general paralysis of the insane the whole course of the disease is comprised within three or four years at most. In general spinal paralysis the post-mortem signs of disease are in the spinal cord and not in the brain; in general paralysis of the insane the reverse of this obtains, the cord being healthy and the brain the seat of disease. Much, no doubt, remains to be done before it is possible to speak positively as to the character of the diseased changes in the cord which are met with in general spinal paralysis; and at present it must suffice to say, that in one case related by Dr. Duchenne there was softening and injection of the anterior columns in the cervical region of the spinal cord, and that in one case which I had the opportunity of examining there was want of proper consistence, not exactly amounting to actual softening, and a perceptible degree of atrophy, in these columns, throughout the whole of their course from the middle of the neck downwards. Whether general spinal paralysis will prove to have that relation to disease of the anterior columns of the cord which locomotor ataxy has to disease of the posterior columns, remains to be seen.

General spinal paralysis blends, no doubt, with other spinal diseases, and its symptoms vary accordingly; but still it occurs with sufficient frequency in the form described by Dr. Duchenne to deserve the position which he assigns to it as an individual malady. There are also relations equally intimate between general spinal paralysis and cerebral maladies, and I am very much disposed to think that the cases in which the mental powers are obviously weakened will be found to be at least as numerous as those typical cases in which these faculties are natural. At the same time it must be borne in mind that in some cases of general spinal paralysis the mind may seem to be weakened, when reality it is not so—that in some cases there may be an air of stupidity, or even fatuity, arising from the slow play of the features, the thickness of the speech, the fumbling of the fingers, and like symptoms, which air has its origin in the paralysed state of the muscles and not in the enfeebled state of "the man behind the mask."

General spinal paralysis cannot be confounded with local Cruvelhier's atrophy, or lead palsy, and it must not be confounded with the general forms of these maladies. In general Cruvelhier's atrophy, as well as in local, the atrophy of the muscles is partial, certain muscles being, as it were, dissected out, and others left untouched, capriciously; in general spinal paralysis the atrophy is *en masse.* In general Cruvelhier's atrophy, what remains of muscle obeys the will and reacts with electricity properly—there is no paralysis; in general spinal paralysis there is true paralysis, and the paralysed muscles have lost much of their electro-contractility. In general lead palsy, also, the history will be sufficient to prevent any confusion as to diagnosis —the paralysis at first electing the extensor muscles of the forearm, the blue line upon the gums, the colic, the constipation, the possibility of lead contamination, and so on.

As regards treatment there is nothing to be said except that it must be conducted upon the same principles as those which apply in analogous cases.

VIII. Hysterical Paraplegia.[1]

Paralysis is certainly entitled to a place among the symptoms of hysteria. Dr. Briquet met with it in 113 out of 430 hysterical patients, its seat being in the four extremities and in the principal muscles of the trunk in 6, in the left arm and leg in 46, in the right arm and leg in 14, in both arms in 5, in the left arm only in 7, in the right arm only in 2, *in both lower limbs in* 18, in the left lower limb in 4, in the feet and hands in 2, in the face in 6, in the larynx in 3, in the diaphragm in 2; and my own experience is more in harmony with these statistics than with the statement of Todd, that the face and tongue escape in hysterical paralysis, that the hemiplegic form of paralysis is less common than the paraplegic, and that "hysterical aphonia" is the form which is most frequently met with.

Hysterical paralysis, so called, is generally met with in persons of a nervous habit of body, and in conjunction with symptoms of an unmistakeably hysterical character. As a diagnostic feature, Todd laid stress on a peculiar expression of countenance, which he denominated *facies hysterica*—an expression characterised by a remarkable depth and prominent fulness, with more or less thickness of the upper lip, and by a peculiar drooping of the upper eyelids; and, as it would seem, with good reason. Often, moreover, there is a definite history of symptoms which clearly come within the category of hysterical phenomena—emotional excitability, globus, plentiful gushes of pale urine, and the rest. In diagnosing hysterical paralysis, however, it is not necessary to trust solely, or even chiefly, to evidence such as this, for the paralysis itself is found to have certain features which in themselves are sufficiently distinctive.

Hysterical paralysis is characterised by the paralysis being more or less incomplete, by a marked degree of numbness being associated with it, and chiefly (according to Dr. Duchenne) by the paralysed muscles, which are not wasted, *having lost their electro-sensibility without losing their electro-contractility*—a loss which, by the way, does not support Sir Benjamin Brodie's opinion that it is the power to will contraction, and not the power of executing the orders of the will, which is at fault in this form of paralysis.

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[1] See also article on Hysteria, p. 315 et seq.

It would also seem to be a peculiarity of hysterical paralysis, as well as of hysterical hyperæsthesia, anæsthesia, and clonic convulsion, to affect the *left* side of the body rather than the right. Thus, M. Briquet found pleurodynia nineteen times, hyperæsthesia and anæsthesia five times, clonic convulsion twice, and paralysis thrice as frequent on the left side as on the right side. He found, indeed, a state of things which presents a contrast to what is met with in rheumatism, neuralgia, pleurisy, pneumonia, and other maladies, in all of which it is the right side of the body which is most prone to suffer.

Very frequently, I believe, hysterical paralysis is preceded by symptoms which come under the head of spinal irritation, and not unfrequently, especially when the upper part of the body is affected, it is ushered in by emotional and other symptoms which may at times deserve to be spoken of as an attack of hysteria.

Hysterical paraplegia agrees in its essential features with other forms of hysterical paralysis. The paralysis is usually incomplete. Numbness of the paralysed parts is a conspicuous phenomenon; as conspicuous, it may be, as the paralysis. The paralysed muscles have lost their electro-sensibility without losing their electro-contractility. The bladder and bowel (as much apparently for want of proper sensibility as from true paralysis) are little under control, if at all; less so, as a rule, than in common paraplegia. The paralysis is often preceded by symptoms of spinal irritation, in the lumbar region especially,—spinal tenderness, pains about the pelvis and in the legs, irritability of the bladder, and the rest; and now and then it is ushered in by some ordinary hysterical disturbance of one kind or other. And where one leg only is affected, there would seem to be, as Todd pointed out, a gait which is not less characteristic than that which is seen in common hemiplegia. In common hemiplegia the trunk in walking is first of all inclined to the sound side, and the whole weight of the body made to rest upon the sound leg, and then the paralysed limb is raised from the ground and thrown forwards by swinging it outwardly; the whole series of movements being very like those which are necessary in walking with a wooden leg. In hysterical paralysis, where one leg only is affected, the paralysed limb, instead of being raised from the ground, as in common hemiplegia, and thrown forward by an outward swing, is dragged directly forward, with the foot trailing on the ground.

The prognosis in hysterical paralysis would always seem to be favourable. Sooner or later, in one way or another, a cure is brought about—most tardily, perhaps, in the paraplegic form of the disorder.

As regards treatment, all that need be said is, that general rules must be followed out, and that, if anything special has to be done, most help will probably be derived from sharp faradization with electrodes which allow the currents to act on the sentient nerves rather than on the muscles—that is, with metal ends rather than with the moistened sponges commonly used. At any rate, sharp practice of this kind has often served to bring about results as sudden and satisfactory as those which have now and then followed the exercise of faith in the power of St. Médard and other kindred agencies.

IX. REFLEX PARAPLEGIA.

Paraplegia is one of the consequences of primary disease in the spinal cord: of this there can be no doubt. Paraplegia may also be the result of disorder or disease beginning at a distance and affecting the cord secondarily—beginning in the urinary and genital organs more especially: of this there can be but little doubt. In the former case the paraplegia is spoken of as centric; in the latter as eccentric or *reflex*.

The chief characteristics of that form of reflex paraplegia which is associated with disease of the urinary organs—*urinary paraplegia,* as it is often called—the commonest and most important of all the forms of reflex paraplegia, as it certainly is, are these; or at any rate these are those upon which Dr. Brown-Séquard, who has paid much attention to this subject, insists. Usually the paralysis is incomplete both as to degree and extent, some muscles being obviously more affected by it than others; usually the paralysis is *not* associated either with tingling or numbness, or anæsthesia; usually the bladder and rectum are only slightly implicated in the paralysis; usually there are changes for the better or worse in the degree of paralysis corresponding to changes for the better or worse in the disease of the urinary organs; usually there is no marked atrophy in the paralysed muscles. Not unfrequently a cure or marked amelioration in the paralytic condition is brought about by the removal of the disease in the urinary organs. Dr. Brown-Séquard indicates these as the chief characteristics of reflex paraplegia connected with disease of the urinary organs, and of other forms of reflex paraplegia as well, the only difference in the description of these latter forms of disease being the substitution for the term "urinary" of the name which indicates the starting-point for the paralysis.

Thus defined, reflex paraplegia differs diametrically from the paraplegia produced by myelitis. In paraplegia from myelitis the paralysis is usually complete, and all the muscles are affected equally: not so in reflex paraplegia. In paraplegia from myelitis the paralysis is associated with tingling, numbness, or anæsthesia: not so in reflex paraplegia. In paraplegia from myelitis paralysis of the bladder and lower bowel is a marked phenomenon: not so in reflex paraplegia.

In paraplegia from myelitis the paralysed muscles are usually atrophied and degenerated: not so in reflex paraplegia. In paraplegia from myelitis cure, or even improvement, is the exception: in reflex paraplegia it is the rule.

It is, indeed, easy enough to find marked differences between paraplegia from myelitis and reflex paraplegia; but the case is far otherwise when a comparison is instituted between paraplegia from spinal congestion and reflex paraplegia. In reflex paraplegia the paralysis is incomplete, and all muscles are not affected equally: in paraplegia from spinal congestion it is so also. In reflex paraplegia the paralysis is not associated with tingling, numbness, or anæsthesia: in paraplegia from spinal congestion it is the same, with the single exception, that there may be at one time or other a trifling degree of tingling at the extreme tips of the fingers or toes. In reflex paraplegia there are fluctuations in the degree of the paralysis: so also in paraplegia from spinal congestion. In reflex paraplegia there is no marked change in the nutrition of the muscles: so also in paralysis from spinal congestion. And, lastly, in reflex paraplegia, as in paraplegia from spinal congestion, a cure is neither an impossible, nor even an improbable, event. As to essential characteristics, indeed, I can find marked differences when reflex paraplegia is compared with paraplegia from myelitis, but none when reflex paraplegia is put in comparison with paraplegia from spinal congestion.

Nor is reflex paraplegia *always* to be distinguished by being obviously *preceded* by eccentric disorder in the urinary organs or elsewhere. It is, indeed, as Sir W. Gull has well pointed out, "not always easy to determine at this point whether symptoms have a central or a peripheral origin. . . . There is, perhaps, no fact to be more insisted upon than the normal dependence of the sympathetic upon the integrity of the spinal system. As a result of this dependence, we learn that dyspepsia, vomiting, constipation, colic, vesical catarrh, prostatic irritation, pains in the joints, and many other peripheral disturbances, may seem to precede the central malady, and to be the cause of it, when in truth they are its effects." And again: "It is no new fact in medicine, that cerebral exhaustion may impair the functions of the cord (especially of the lower segments), and give rise to precisely those symptoms which have been set down as pathognomic of urinary paraplegia."

Dr. Brown-Séquard has taken a very different view of reflex paraplegia to that which is here taken. He regards this disorder as due, not to spinal congestion, but to a state of the circulation diametrically opposed to this. He believes that a state of irritation, commencing eccentrically, is propagated along the vaso-motor nerves, of which the result is, primarily, contraction of blood-vessels in, and, secondarily, exclusion of the due amount of blood from, one or more of the three parts following—the spinal cord, the nerves proceeding to or coming from the cord, the muscles. He believes that the proper activity of the nervous tissue or muscle is starved into paralysis for want of blood; and he founds this view on the fact that a state of irritation in the vaso-motor nerves may proceed from a distant point and produce contraction of the vessels, and upon the fact that traces of organic disease are wanting after death in many cases of reflex paraplegia. The argument, indeed, is all but as conclusive as it is masterly and original. The same evidence, however, admits of a very different construction, and that even without anything like special pleading. It is, no doubt, true enough that a state of irritation in vaso-motor nerves may lead to contraction in blood-vessels, and thereby exclude a due amount of blood from the part to which these vessels belong; but it is not less certain that the same state of irritation carried beyond a given degree, either in time or in intensity, may, by paralysing the vaso-motor nerves, lead to relaxation of vessels, and, thereby, to the admission into them of an undue amount of blood.

Moreover, it may also be assumed, as a thing by no means improbable, that the contraction of the coats of the relaxed and paralysed vessels in rigor mortis may prevent any marked traces of such vascular engorgement being met with after death; at any rate it is impossible to infer, from the absence of such traces of congestion after death, that there was no such congestion during life. In itself, indeed, the evidence adduced by Dr. Brown-Séquard in favour of his theory of reflex paraplegia is insufficient to decide whether his view or that which I venture to put in opposition to it is the correct one, for in reality it may be used equally in support of either view. And certainly it would seem to be a collateral objection to the view which connects reflex paraplegia with a state of capillary contraction and comparative bloodlessness brought about by irritation in vaso-motor nerves, that in states where the whole nervous system is in a state of great irritation, as in tetanus, and in the state specifically designated spinal irritation, and where it may be assumed that the vaso-motor nerves participate in this state of irritation, and produce vascular contraction and comparative bloodlessness in the spinal cord and elsewhere, that paraplegia or any form of paralysis is precisely the symptom which is not present. Moreover, Sir W. Gull makes some remarks on urinary paraplegia which have an important collateral bearing on the subject in hand, as tending in no ordinary degree to support the conclusion to which all the previous considerations tend: "If," he says, "we regard the nature of the urinary disease which most commonly leads to paraplegia, we shall find that it is an inflammation, either in the prostate, bladder, or kidneys; and we shall also find, that it is only after chronic inflammation has lasted a long time that the paraplegic weakness supervenes. It is in just those cases where there is most irritation, and but little inflammation, that paraplegia does not occur. Uric acid and oxalate of lime calculi may cause hæmaturia and any amount of irritation, but unless *suppurative* inflammation set in, paraplegia is not produced. A review of all the recorded cases of urinary paraplegia will show that it is the *inflammatory* condition of the urinary organs which leads to paralysis, and not one of irritation."

In speaking in this manner, however, I do not wish to confound reflex paraplegia with spinal congestion. On the contrary, the more I see of practice the more I am disposed to think that there is a reflex variety, not only in paraplegia from spinal congestion, but in every form of paraplegia; that, in fact, the causes at work in producing all spinal maladies are reflex in their character as well as centric,—reflex, it may be, rather than centric.

If the true view of reflex paraplegia be the one which is here taken, it follows that the treatment of that form of this disorder which is defined by Dr. Brown-Séquard will be substantially the same as the treatment of paraplegia from spinal congestion, and not that which has been recommended on the supposition that the spinal cord is starved for want of blood in consequence of its vessels being kept in a state of contraction by irritation of the vaso-motor nerves. Nay, even the necessity to treat eccentric disorder or disease in the urinary organs or elsewhere can scarcely be considered a peculiar feature in the treatment of reflex paraplegia; for, in fact, it is always an essential part of any sound plan of treatment in any disease of the spinal cord, whether originating in the cord or at a distance from the cord, to make a point of doing everything to remove or mitigate any eccentric malady. It is always necessary to do this, because an eccentric malady, whether primary or secondary to the spinal disorder, or whether having no other than a purely accidental relation to this disorder, invariably *reacts* prejudicially upon the cord. This eccentric malady must of course be dealt with on general principles, this thing or that being done according as irritation or inflammation may happen to be the predominating condition. In urinary paraplegia, for example, it is very possible that the local application of opium or belladonna to the urethra, as recommended by Dr. Brown-Séquard, may be of much use; this is very possible on any hypothesis: but with respect to the frequent introduction of catheters, with a view to relieve irritation, I think it is difficult to come to a different conclusion to that which Sir W. Gull has arrived at. "This course," says this able physician, "is not unattended with danger. There is no part of the treatment which calls for more discrimination. The diseased textures and veins about the neck of the bladder are so prone to suppuration, that the catheter is often a fatal weapon. The few scattered instances, such as that recorded by Dr. Graves, where immediate good effects have followed, have had undue influence towards promoting mechanical interference. Carefully considered, they do not warrant the inference drawn from them. If the urinary passages are so contracted that the bladder cannot empty itself, the catheter is obviously required; but it must be simply prescribed on these grounds. The rule for its use is the same as in the treatment of the aural passages, when the middle ear is diseased. If there be a free exit for the excretions, the less mechanical interference the better. As meddlesome midwifery is bad, so is the meddlesome employment of the catheter in urinary paraplegia. Cases might be quoted where a fatal issue has been induced by the meddlesome interference with a diseased bladder, under the hope of removing some hypothetical cause of reflex irritation."

X. INFANTILE PARALYSIS.

This disorder, to which attention seems to have been directed first of all by Underwood, Marshall Hall, and Kennedy, is the *paralysie (dite essentielle) de l'enfance* of several French writers. It attacks children indiscriminately, without any regard to sex, between the age of six months and two years, at the time of the first dentition more especially: and it is the grand source of shrivelled, half-dead limbs, club-feet, and other sad deformities.

Mr. William Adams, who has had ample opportunities for becoming practically acquainted with the history of infantile paralysis, and whose account of this disorder is more to the point than any other with which I am acquainted, indicates these as the most trustworthy characteristics:—1. The paralysis is usually partial, single muscles or groups of muscles only being affected. 2. The sensation in the paralysed parts is usually perfect, or all but perfect. 3. The bladder and lower bowel are usually not distinctly implicated in the paralysis. 4. The paralysed muscles are at no time rigid. 5. Great improvement or complete recovery is the rule, and not the exception. 6. The paralysis is usually neither accompanied nor preceded by "head symptoms."

The onset of the disorder is generally sudden and unexpected. The child is put to bed well, and in the morning it is found to be paralysed. Or the paralysis may be grafted upon some marked febrile disorder, as gastric or remittent fever, measles, or typhus; or upon some other malady, as hooping-cough or pneumonia. In some cases there may be transitory and trifling feverishness at first, but fever is certainly no essential accompaniment at any time. Now and then, but only in exceptional cases, the disorder may be ushered in by convulsions or drowsiness.

The paralysis has usually a wider range at first than that which it takes afterwards; in other words, the paralysis is more or less general at first, and more or less localised afterwards. Thus it is a common thing for all the limbs to be attacked and for only one leg to remain paralysed, or, rather, to remain partially paralysed, for there is a certain degree of recovery in certain muscles, even in the worst cases. It is the constant rule, indeed, for recovery to be slower in the legs than in the arms, and in certain muscles than in others. Usually the disease does not mount high enough to paralyse muscles whose nerves are given off above the true limits of the spinal cord. There is certainly no loss of sensation in infantile paralysis. On the contrary, as Dr. West remarks in his admirable treatise on the diseases of infancy and childhood, "sensation in the affected limb appears to be exalted when the paralysis is recent, the degree of hyperæsthesia in the early stage being in such cases proportionate to the loss of power which afterwards is apparent." Moreover Dr. West proceeds to say, "In some instances the exaggerated sensibility continues for several weeks, though this is unusual; and when this is the case, the leg being the seat of the affection, and the paralysis incomplete, the existence of hip-joint disease may very likely be suspected. In such a case the child bears all its weight on the healthy limb, turns the foot of the affected side inwards when walking, and stands with the toes of that foot resting on the dorsum of the foot of the healthy side. Still it will usually be found that the exaggerated sensibility of the paralysed limb varies greatly at different times, while that extreme increase of suffering produced in cases of hip-joint disease on striking the head of the femur against the acetabulum by a blow upon the heel, and the fixed pain in the knee of the affected side, so characteristic of diseases of the hip-joint, are absent; and these points of difference will enable you to distinguish between the two affections. One other important means of diagnosis is furnished by the presence or absence of an increased temperature over the suspected joint, the value of which means in determining the presence or absence of inflammation about any particular spot is dwelt upon by Mr. Hilton in his lectures delivered recently at the College of Surgeons."

The peculiarities of infantile paralysis, so thinks Mr. Adams, point to a special pathology which has yet to be made out satisfactorily. As it seems to me, however, these peculiarities, instead of showing, as Mr. Adams believes, that infantile paralysis is unlike paralysis in adults, only show a close analogy to, if not an actual identity with, the paralysis which has been seen to result from spinal congestion. In infantile paralysis the paralysis is partial: in paralysis from spinal congestion it is the same. In infantile paralysis sensation is exaggerated rather than dulled in the paralysed parts: in paralysis from spinal congestion it is the same. In infantile paralysis the bladder and lower bowel are obedient to the will: so also in paralysis from spinal congestion. In infantile paralysis the paralysed muscles are limber, not rigid: so also in paralysis from spinal congestion. In infantile paralysis recovery more or less complete is the rule rather than the exception: so also, and very much in the same order, in paralysis from spinal congestion. In infantile paralysis "head symptoms" are exceptional phenomena at any time: so also in the paralysis from spinal congestion. Neither do I know of anything to invalidate the conclusion which those resemblances would seem almost to necessitate—that infantile paralysis, as defined by Mr. Adams, is nothing more than paralysis from spinal congestion.

Moreover, this conclusion is not discredited by the disclosures of morbid anatomy. There were no traces of organic disease either in the spinal cord or brain or nerves in the four cases of genuine infantile paralysis which were examined after death by MM. Barthez and Rilliet, Dr. Fliess, and Mr. Adams, all four most competent observers.

The evidence supplied by these cases is indeed purely negative. Nor is evidence more positive to be found in the two cases examined after death by M. Laborde, the writer of a very able treatise on infantile paralysis recently published. In these two cases, without doubt, there were certain organic changes in the spinal cord and in some of its nerves, but these changes are plainly not essential to infantile paralysis as defined alike by M. Laborde and Mr. Adams; for the simple fact is, that the clinical history of these cases is not clearly that of infantile paralysis so defined. In a word, there is nothing in the scanty contributions of the dead-house to show that the very closest relations may not exist between the disorder under consideration and spinal congestion.

The duration of infantile paralysis is very variable. It may pass off in a few days, or even a few hours: it is more likely to occupy several weeks or months in this process of improvement. Improvement, to a greater or less degree, is indeed the rule, and not the exception; and it may even be said that the cases which stop far short of recovery are by no means common. Mr. Adams says, "It is generally supposed that, unless recovery takes place within a few months, the paralysis is persistent through life; but I have seen many cases in which improvement has proceeded, to a very useful extent, several years after the seizure;" and to the truth of this remark my own experience bears ample testimony. Indeed, I should say from what I have seen, that if the paralysed muscles retain their electro-contractility and electro-sensibility, and so show that they have not passed into that state of fatty degeneration into which they always tend to pass eventually, there appears to be scarcely any limit to the time in which improvement, and even complete recovery, is possible.

The groups of muscles most frequently affected in infantile paralysis, according to Mr. Adams, are—1. The muscles of the anterior part of the leg, forming the extensors of the toes and the flexors of the foot; 2. The extensors and supinators of the hand, these muscles being always affected together; and 3. The extensors of the leg, and with them generally the muscles of the foot, as in the first group. When single muscles are affected, the most likely to suffer are these:—1. The extensor longus digitorum of the toes; 2. The tibialis anticus; 3. The deltoid; and 4. The sterno-mastoid.

The deformities produced by infantile paralysis are most frequently met with in the feet and legs, because these are the parts most frequently affected; and the particular kind of deformity varies, of course, with the muscles involved in the paralysis.

"The most frequent kind," says Mr. Adams, "is that of (1) talipes equinus; and the other deformities occur in the following order,—(2) equino-varus; (3) equino-valgus; (4) calcaneus, or calcaneo-valgus of one foot is generally found with equino-valgus of the other."

Mr. Adams is of opinion that the great cause of the deformities which are met with in infantile paralysis is the "adapted atrophy" of Sir James Paget, this change taking place chiefly in the opponents of the muscles which have suffered from paralysis. If, for example, the anterior muscles of the leg are paralysed, the anterior portion of the foot drops, and the heel is raised, not by active contraction of the posterior muscles—for division or paralysis of one set of muscles does not excite active contraction in the opponent muscles—but in consequence of the position assumed by the foot from its mechanical relations with the leg. Another cause of deformity is obviously atrophy and actual or comparative arrest of development in the paralysed muscles; for, unless the paralysis soon passes off, it is plain that the muscles will not only waste, but be left behind in the rapid process of development which is everywhere at work in a young and growing child. Mr. Adams is also of opinion that the early and late rigidity of Todd and true spasm have very little to do in causing the deformities in question: and so it may be in the deformities connected with that form of paralysis to which he restricts the term infantile—that form which is undoubtedly the common variety of infantile paralysis, and which, as it would seem, is dependent on spinal congestion. It is very certain, however, that infants and children are liable to more than one form of paralysis, and that there are deformities associated with rigid as well as with flaccid muscles. It is very certain that this rigidity may be either "early" or "late," as distinguished by Todd, or even still more decidedly spasmodic than that form which is called "early rigidity." In a word, infantile paralysis is a designation as little to be defended as would be the term adult paralysis; for on inquiry it is found that in children, as in adults, there is more than one form of paralysis, and that all the forms which may happen in adults may be repeated in children. The form of paralysis which has been described as infantile is unquestionably the commonest, and the other forms are so uncommon as to be little more than exceptional; and this, in fact, is all that can be said to justify the notion that infantile paralysis is a definite disorder of the spinal cord peculiar to infants.

The treatment of the deformities, especially of club-foot, resulting from the so-called infantile paralysis, is a subject of much practical interest and difficulty. Mr. Adams says:—"The probability of benefit in such cases by any surgical procedure seems scarcely ever to be entertained. The existence of paralysis is supposed to contra-indicate any surgical interference; but, from these apparently hopeless and essentially incurable cases some of the most striking and most valuable results of surgery are obtained by a combination of surgical and mechanical treatment. Mechanical aid, alone, is frequently sought from the instrument-maker, but his art is powerless when any considerable amount of deformity exists; and it is only by a scientific combination of surgical and mechanical skill that much good can be effected. In all these cases the treatment essentially consists in the removal of existing deformities by tenotomy and mechanical means, and a subsequent compensation for the existing paralysis by mechanical support, varying in different cases according to the extent of the paralysis."

And no doubt very satisfactory results are obtained by those means. At the same time it is certain that in many cases very satisfactory results may be obtained without tenotomy, and without apparatus, by means used with the view of bringing back power into the paralysed muscles—electricity,[1] movements of various kinds, shampooings, and others; and my own experience has convinced me that this fact is not yet sufficiently recognised and acted upon in practice. That in many cases neither tenotomy nor apparatus can be dispensed with, I fully believe: that in all cases the electrical and gymnastical parts of the treatment are of primary rather than of merely secondary importance I am every day more and more convinced, because every day I meet with instances of muscles which I should once have looked upon as hopelessly paralysed being resuscitated by those means. Indeed, I cannot but think that so long as institutions especially set apart for orthopædic purposes are wanting in properly furnished electrical rooms and gymnasiums, there must be in some essential points a necessity for a great reformation in orthopædic practice.

XI. SPINAL HÆMORRHAGE.

Blood may be effused into the substance of the cord between the arachnoid and pia mater, into the sac of the arachnoid, between the dura mater and arachnoid, or between the dura mater and the osseous canal—anywhere in or about the spinal cord, in fact. Hæmorrhage in the substance of the cord, the *hæmatomyélie* of Ollivier, may be a consequence of myelitis, the blood-vessels breaking up in the softening of the cord, and so allowing the blood to escape. It was so in the acute case which I took as my text when speaking of myelitis, for here the blood was collected at one point in the softened nerve matter to an extent which at first sight suggested the idea of hæmorrhage into the cord rather than that of myelitis. Hæmorrhage under or upon the spinal membranes, the *hæmatorachis* of Ollivier, may be a consequence of cerebral hæmorrhage, the blood overflowing from the cranial into the spinal cavity, and perhaps mixing with the spinal fluid; or it may result from spinal congestion, spinal meningitis, myelitis, tetanus, hydrophobia, and certain other maladies. All these cases, however, are so uncommon as to be little more than exceptional. In fact, hæmorrhage either into the substance of the cord, or under or above the spinal membranes,—except as the result of some accidental injury to the spine, as in death by hanging, or in cases of still-birth where it has been necessary to employ much force to bring about the delivery,—is, to say the least, a very uncommon affection.

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[1] There are certain forms of paralysis in which the paralysed muscles do not react to the most powerful induced electric currents, but react energetically to a galvanic current of low tension, slowly interrupted (the *labile current* of Remak). The diagnostic and therapeutic bearings of this fact have yet to be worked out, but so far the therapeutic promise is good. The phenomenon in question has been already observed in several very different cases—in facial palsy (first noted by Baierlacher), in certain cases of infantile paralysis (discovered by J. Netten Radcliffe, of London, and Hammond, of New York, independently of each other), in certain cases of local palsy, *e.g.* palsy of the extensors of the fore-arm and of other muscles, from lead-poisoning (Bruckner and J. N. Radcliffe), in paralysis of the deltoid, not from lead (J. N. Radcliffe), in certain cases of muscular atrophy (J. N. Radcliffe), and in paralysis from traumatic injury of a nerve (Bruckner).

The symptoms of spinal hæmorrhage are by no means clearly marked. Sudden and acute pain in the spine at the seat of the effusion, and sudden paralysis and loss of sensation, more or less complete, in the parts below this point, appear to be the chief symptoms where extensive hæmorrhage has taken place into the substance of the cord. Sudden and acute pain in the spine would also seem to be a prominent symptom in hæmorrhage below or above the spinal membranes, but not sudden paralysis and anæsthesia. In this latter case, indeed, instead of paralysis there have been some convulsive or spasmodic symptoms, and instead of anæsthesia some hyperæsthesia. In some cases, as in one quoted by Dr. Copland, the pain may be not in the back, but at a distance from the back; and in other cases, and this not unfrequently, pain may be greatly masked by the shock of the accident which has caused the hæmorrhage, or by the shock attendant upon the laceration of the spinal cord by the effused blood. When the hæmorrhage is in the medulla oblongata, and high up in the cord, the symptoms may be rather like those of epilepsy than anything else—loss of consciousness, convulsion more or less general, choking noises, and the rest—and this equally whether the blood is effused into the substance of the cord or around it: and this fact suggests the possibility, to say the least, that the convulsive or spasmodic symptoms, which have by some writers (on what to me seem to be insufficient grounds) been supposed to distinguish hæmorrhage under or above the spinal membranes from hæmorrhage into the substance of the cord, may in reality be due to irritation transmitted to the medulla oblongata and upper part of the cord, and not to irritation acting upon the membrane or membranes. Moreover, when the hæmorrhage is high up in the cord, priapism and distress of breathing are found to figure conspicuously among the symptoms, as they do also in other cases where this part of the cord is damaged by disease or injury. In a few instances, the symptoms of spinal hæmorrhage are preceded by symptoms indicative of spinal congestion, or inflammation, or irritation.

Remains of old apoplectic cysts, similar to those so often found in the brain, have been met with in the spinal cord, even in the medulla oblongata and upper part of the cervical region; but these signs of partial recovery are, to say the least, exceedingly exceptional. Indeed the mischief done by the hæmorrhage is generally not only irreparable, but very speedily fatal, and that too in spite of everything that can be done to promote recovery.

XII. NON-INFLAMMATORY SOFTENING.

Two well-marked varieties of softening of the spinal cord are detected by the naked eye—the red and the white. In both varieties the microscope brings to light broken down nerve-tissue mixed up with a number of bodies known as *granule masses*—large bodies, whose principal constituent is fat, black-looking, from not transmitting light, and somewhat like mulberries, from being built up of a number of round bodies or granules. "It was once thought," said Dr. Wilks, "that these masses denoted inflammation. But you find them in any degenerating part, as a decaying strumous gland, or a cancerous tumour, or a phthisical lung: and the question of their formation in the brain or cord is not yet answered; whether they originate in inflammatory cells, or are the natural cells of the nerve-structure degenerated. In some you may still see a wall and a nucleus, which points to the former opinion as the more correct." The red variety of softening is often in parts yellow rather than red: the redness being due to increased vascularity or effused blood-corpuscles, one or both; the yellowness to the presence of fibrillated tissue, nucleated fibre, pus-corpuscles, or some other form of distinctly inflammatory product. In a word, there can be no doubt of the inflammatory origin of the red variety of softening. In the white variety of softening, on the other hand, there are generally an athermatous state of the vessels and other signs of true degeneration, the vascularity is evidently diminished, and there is an absence of those distinctly inflammatory products which have just been enumerated. It would seem, indeed, that the white variety of softening differs essentially from the red, in that, instead of being the result of inflammation, it is brought about by the parts being starved and atrophied for want of blood. With respect to the reality of these differences between these two varieties of softening there appears to be little or no reason for doubt: at the same time it must not be forgotten that it is not always easy to draw the line between these two varieties, and that they both may exist together in the same cord.

The symptoms of non-inflammatory softening would seem to be identical with those of the more chronic forms of myelitis. The more tardy the development of these symptoms, and the older the patient in years or in constitution, the more likely is the case to be one of non-inflammatory softening: and this is all that can be said in the matter of diagnosis. Practically, however, this want of definiteness is of no moment; for in the chronic form of myelitis the degenerative process has more to do in bringing about the diseased changes in the cord than the inflammatory, and more to do also in supplying the indications for treatment. Nay, it may even be held that the same remark applies to some extent to the more acute forms of myelitis as well as to the more chronic, for it is with the ruin rapidly produced by the inflammation rather than with the inflammation itself that the practitioner in medicine has to cope almost, if not altogether, from the very onset of the disease.

XIII. INDURATION.

Like the opposite condition of softening, induration (sclerosis) of the spinal cord is one of the consequences of myelitis, chronic or acute, of the chronic form more especially. Induration of the cord is generally associated with atrophy—atrophy often more marked in the white matter than in the grey—and with a condition so curiously bloodless that a section is not unlike that of white of egg boiled hard. In its highest degree the cord may have a leather-like or fibro-cartilaginous hardness and consistency. Induration is a much less common change than softening: it has no symptoms by which it can be distinguished from softening: and it is often met with when it was not expected, and under very different circumstances, as after acute myelitis on the one hand, or after long-standing epileptic disease on the other.

XIV. ATROPHY AND HYPERTROPHY.

Atrophy of the spinal cord, like atrophy of the brain, is one of the changes which must be looked upon as natural to old age. In elderly persons, indeed, the cord becomes shorter and narrower and firmer, the spinal fluid increases in quantity, so as to fill the space left vacant by the shrunken cord, and the spinal nerves are sensibly wasted at both their roots. All this has been abundantly proved by Chaussard, Ollivier, and others. Atrophy, more or less general, is also associated with many forms of paralysis in which the cord has been long left in a state of comparative functional inactivity; and local atrophy is one of the consequences of tumour, displaced vertebræ, or anything which exercises pressure upon the cord. Of partial forms of atrophy resulting from disease, the only one about which there is any certain knowledge is that which is associated with the disease called locomotor ataxy—namely, atrophy of the posterior columns; and about this form enough has already been said in a separate article.

In a few instances the spinal cord has been found to be so much enlarged, apparently by a true hypertrophy of its natural tissues, as to occupy the whole space of the vertebral canal; but most generally what seems to be hypertrophy at first sight is due, chiefly at least, to congestive swelling and œdema. True hypertrophy has been met with in the fœtus: it occurs mostly in children: and it presents, so far as is known, no symptoms by which it can be recognised. Hypertrophy of the brain is a very uncommon affection, but it is common as compared with hypertrophy of the spinal cord.

XV. TUMOUR, ETC.

"Tubercle and cancer," says Rokitansky, "are frequent in the brain, unfrequent in the spinal cord. Tubercle I have observed only in combination with other advanced tuberculoses. Its principal seat is the cervical or lumbar portion of the cord, where it sometimes occupies the white fibres, sometimes the grey substance. As in the brain, it leads to inflammation (red softening) and to yellow softening. I have never seen a tuberculous cavity in the cord. Sometimes several tubercles are grouped together, none exceeding the size of millet or hempseed; at other times only one exists, which is of large dimensions, equalling a pea or a bean. Exclusively of several cases of circumscribed callous induration of the white columns, as to the cancerous nature of which I am still in doubt, I have met with but one case of cancer of the cord. It was a solitary nodule of medullary cancer. Ollivier mentions several examples of diffused carcinomatous growths, as well as of the so-called colloid cancer. Among the entozoa I have repeatedly seen the cystocercus in the cervical portion of the spinal marrow. The acephalocyst sacs, as far as has been observed, have no connexion with the cord; their nidus is even outside the dura mater. In one case the cyst forced its way into the cavity of the arachnoid."

Nor are exostoses, cartilaginous growths, or aneurisms frequently met with in positions which can exercise pressure upon the spinal cord. Cartilaginous growths, or rather bony plates, it is true, are not unfrequently met with in the visceral arachnoid of the cord—a condition which appears to be rarely met with in the brain; but these growths or plates can scarcely be brought under the head of tumours. Except, perhaps, in connexion with scrofulous disease of the vertebræ, the pia mater of the cord is not the seat of tuberculous deposits; and here again is another point of difference between the pathological history of the spinal cord and the brain, for it is a well-known fact that the pia mater of the brain is a favourite seat of these deposits.

The symptoms produced by tumour vary greatly. Neuralgic pain in the back, over the seat of the tumour, appears to be an almost constant symptom. "Pain," says Dr. Reynolds, "is more marked in cases of carcinoma than of tubercle." If a particular nerve be irritated by the tumour, there may be pain, tingling, or some other anomalous sensation in the part or parts supplied by its sentient fibres, or some morbid form of contraction in the muscles supplied by its motor fibres. If a particular nerve be pressed upon more decidedly by the tumour, there may be local anæsthesia, or paralysis instead of morbid sensations or muscular contractions. It is but seldom, however, that these symptoms of irritation or pressure are so strictly localised; and, in fact, the presence of the tumour is made known usually only by more general symptoms of irritation, or compression, or inflammation, which, instead of being in any way pathognomic of tumour, may arise from many other causes. "There is, indeed," as Sir W. Gull says, "no symptom, or single group of symptoms, which, taken alone, can serve as a secure basis for diagnosis." Tuberculous or carcinomatous deposits elsewhere, with signs of the peculiar dyscrasia of tubercle or cancer, aneurism elsewhere, nodes elsewhere, may help to a diagnosis by showing that symptoms which appear to point to a tumour may have such a cause, and at the same time may supply some information as to the special character of the tumour; but this possibility of help in diagnosis is too remote to be of much practical value, if any. It may be supposed that any scrofulous deposit in the cord is more likely to occur in children, and any cancerous growth in older persons; but even this rule has too many exceptions to make it of much use.

XVI. Concussion.

Concussion of the spinal cord, like concussion of the brain, is the result of a fall from a height, a blow on the back, or some other accident, and its symptoms vary with the intensity of the shock. Sudden paralysis and loss of sensation, more or less complete, with some inability to pass water or to prevent the escape of flatus or fæces, are the more special symptoms. Sudden and marked failure in the circulation and respiration, as shown by pallor, feebleness of the pulse, diminished temperature, slow and shallow breathing, and other signs of common shock, are also associated with the more special symptoms. Great pain along the spine or in some part of the spine has been considered as one of the symptoms of spinal concussion; but neither pain nor spasm are met with in the cases which I have examined; and Dr. Reynolds comes to the same conclusion, for speaking of these cases, he says, "There is in them neither marked pain nor spasm." Indeed, in the majority of cases the patient is obviously rendered incapable of experiencing pain by the fact of being *stunned.*

The symptoms of spinal concussion not unfrequently issue in those of spinal congestion, or myelitis, or spinal meningitis, or else death without any signs of reaction may be the result. Often, without passing into any definite disease, the cord, even after what might at first seem to be only a slight degree of concussion, may not recover its former power perfectly, the patient ever afterwards being weak in many respects, especially in his legs and bladder. Indeed, concussion of the spine sufficiently severe to produce at the time any marked degree of paralysis in the limbs and bladder and lower bowel, with loss of sensation, is certainly a very grave matter, and it may be questioned whether in such a case recovery is ever more than partial.

The appearances after death may present nothing unnatural, or they may be those of hæmorrhage more or less extensive. It is very possible that the cases in which severe pain in the back was a symptom would prove, if *all* the *facts* were fully known, to be cases in which the symptoms of concussion were mixed up with those of hæmorrhage: at any rate, there was hæmorrhage in one case of spinal concussion in which pain in the spine was a conspicuous symptom, which case came under my own notice not long ago. In fatal cases, in which the reaction after the concussion has issued in inflammatory and other changes in the cord, these changes will be met with after death; and if fracture or dislocation of the vertebræ was produced at the time of the concussion, the evidence of such injury will of course not be wanting.

XVII. COMPRESSION.

When the spinal cord is compressed by a dislocated or fractured vertebra, by a tumour, by a bullet, or in any other way, the symptoms will of course vary with the seat and degree of compression. The symptoms will, in fact, be as variable—for they will be the same—as those which are produced by experimental division of the parts compressed, and about which more than is convenient had to be said in the preliminary remarks. All, therefore, that is necessary here is to refer to those preliminary remarks for the information which may help to make the symptoms of compression intelligible, and, in passing, to express a hope that trephining or other operative procedures which have been recommended and practised in certain cases of spinal compression may not be altogether unjustifiable.

XVIII. CARIES OF THE VERTEBRAL COLUMN.

This disease is usually limited to the bodies of the vertebræ and to the intervertebral substances, but sometimes it extends backwards to the arches and processes of the vertebræ as well. It commences, very generally, in the middle dorsal region, and, as generally, it does not extend beyond this region; but there is no part of the spinal column in which it may not begin, or to which it may not extend: it invariably, when sufficiently advanced, gives rise to "angular curvature," or projection directly backwards of the diseased part of the spine, this deformity being due to the way in which the thinned and diseased bodies of the vertebræ become crushed in under the weight of the upper part of the body. In the great majority of cases caries of the vertebræ is an unmistakably strumous affection, being neither more nor less than tuberculous infiltration of the bodies of the vertebræ; and the changes in the bone are due to the melting down of this deposit rather than to any strictly inflammatory process.

The earlier symptoms of caries of the vertebræ are not at all well marked. Of these the most conspicuous are—weakness in the back, generally in the dorsal region, with aching or pain, more or less severe, in the weak part, causing a disposition to lean forward and to use the arms as props; some prominence of the spinous processes of the weak and painful part of the spine, with some puffiness of the overlying skin; a feeling of undue heat, or even burning, in the weak and painful and prominent part, which is not felt in other parts of the spine, when a sponge soaked in moderately warm water is passed down the spine; and a state of tenderness on pressure or percussion, which is equally restricted to the same weak and painful and prominent part. Afterwards, when the disease is more advanced, there are more marked symptoms, namely these:—unmistakable "angular curvature," the formation of abscess, slight hectic in the evening, a feeling of constriction around the waist, it may be, and still later, more or less paralysis of the legs, more or less loss of control over the bladder and bowel, and other symptoms indicative of secondary myelitis or spinal meningitis. Abscess may be one of the earlier symptoms preceding any obvious deformity, or it may not occur at all. In fact, abscess appears to be a symptom of strumous disease of the vertebræ exclusively, and not of the non-strumous variety of caries. When it does occur, which is certainly in the great majority of cases, there is usually some diminution of pain and other evidences of irritation. When it does occur, as is well known, it generally makes its appearance at a distance from the diseased vertebræ, most commonly as "psoas abscess" in the groin, but by no means exclusively in this form and locality. It is seldom that the spinal cord becomes *compressed* by the giving way of the bodies of the vertebræ in the progress of the disease: but sooner or later it almost constantly happens that the cord or its membranes opposite the diseased vertebræ become the seat of inflammatory changes, which changes, rather than the drain from an abscess, are indeed the reason why, in so many cases, sooner or later, caries of the vertebræ proves to be destructive to life.

The diagnosis between "angular curvature" from caries of the spine, and the curvatures forward, backward, and sideways, without other structural changes in the vertebral column than those of simple adaptation to the altered position, is not very difficult. These latter curvatures, in fact, want all the special and grave features which have been indicated as characterising the former. Nor yet is the diagnosis difficult between "angular curvature" in its earliest stage and spinal irritation, with which it is sure to be associated, and with which there is certainly no small danger of its being confounded. This topic has been already touched upon when speaking of spinal irritation, and here it is enough to say that the occurrence of the symptoms which are present in the beginning of caries of the vertebræ (which are no other than those which may belong to simple spinal irritation), in children or youths of a manifestly scrofulous habit—at an age, that is to say, and in a habit, in which symptoms of simple spinal irritation are not likely to be met with—are sufficient to do more than create a bald suspicion of the existence of disease of the vertebral column.

The prognosis of caries of vertebræ is always bad enough. A humpback is the best result to be hoped for. The end to be aimed at in treatment is, of course, to promote anchylosis of the diseased bones of the vertebræ by allowing them to fall together—by favouring, that is to say, the deformity which is inevitable by letting the back bend and not by trying to prevent it by keeping the back straight,—and to keep up the strength in every way. But these are matters which I cannot touch upon without trespassing upon the domains of surgery, and I therefore leave them to those who are better able, and whose right it is, to deal with them.

XIX. Spina Bifida, etc.

The commonest congenital affection to which the spinal cord is liable is dropsy, or hydrorachis, and of this dropsy *spina bifida* is the variety most frequently met with, and of most practical interest. The spine is bifid in this disorder from the non-development or separation of the spinal processes and laminæ, and the consequence of this malformation is that an opening is left through which, very often, the dropsical fluid presses outwards, and distends in so doing the integuments and subjacent tissues into an hernial tumour. Very generally congenital hydrocephalus is associated with congenital hydrorachis. The fluid in hydrorachis is precisely of the same constitution and character as that which is met with in hydrocephalus: it varies in quantity from a few ounces to several pints: it accumulates between the arachnoid and pia mater, in the arachnoid sac, in the central canal of the cord, and even outside the dura mater, sometimes in one place, sometimes in another, sometimes in more places than one. The hernial tumour into which this dropsical fluid bulges outwardly varies greatly both in position and size, and in the condition of its coverings: it is almost invariably met with in the lumbar region, but it may be in any region: it is usually of the size of a walnut or orange, but it may be as large as a child's head, or even larger: it may be single or multiple: its bulk may vary considerably under different circumstances, or not at all, becoming, if it vary, fuller and more tense if the position of the child be made such as to cause the fluid to flow into it, emptier and flaccid if this position be altered so that this fluid may run out of it, or if pressure be made upon it so as to bring about the same result: it may swell during expiration and fall during inspiration: it may present distinct fluctuation or none at all; and the skin over it may be sound, thickened, inflamed, ulcerated, gangrenous, covered with tufts of hair, and so on. The dura mater and its lining of arachnoid membrane always enter into the composition of the coverings of the tumour, and these are the only constant elements in these coverings. In the lumbar region, the cord and its nerves, which are generally rudimentary, are out of the tumour altogether: in the cervical and upper dorsal region, on the contrary, it is no uncommon thing for the cord and its nerves to be adherent to the walls of the tumour.

In spina bifida the lower limbs are generally paralysed as well as the bladder and lower bowel, and not unfrequently there is, in addition to the spinal deformity, deficiency of the abdominal walls, hernia of the bladder, imperforate anus, &c. But few cases recover, or even improve, death happening generally at an early period either in convulsions or from spinal inflammation, the immediate cause often being the bursting of the tumour: still there are cases on record in which life has been prolonged—and this too with tumours of no small size—not only for a few months, but for 17, 18, 19, 21, and even 50 years.

There is little to be done for the relief of spinal bifida. Pressure on the tumour by means of an air-pad and suitable bandages can do no harm; and occasional punctures with a grooved needle, as recommended by Sir Astley Cooper, may be a justifiable measure. Even cures have resulted from a combination of these punctures with pressure. "All the plans of treatment," says Mr. Erichsen, "by which the tumour is opened and air allowed to enter it, are fraught with danger, and will, I believe, inevitably be followed by the death of the child from inflammation of the meninges of the cord and convulsions."

There are several other congenital affections of the cord, of which the best account is still to be found in the classical pages of Ollivier. The cord may be entirely absent (*amyélie*); or it may be imperfect (*atelomyelie*). Of the imperfect forms of cord there are several varieties. The upper part may be wanting, as in anencephalous and acephalous monsters. The cord may be bifurcated at one extremity or the other, at the upper extremity in monsters with two heads and one body, at the lower extremity in monsters with one head and two bodies. It may be double. It may vary greatly in dimensions, being larger or smaller, longer or shorter than natural—longer, for example, in monsters with tails, shorter in monsters of a contrary sort. It may, as in one form of hydrorachis, be little more than a long bag in consequence of the distension of the central canal of the cord with the dropsical fluid. Or it may be discoloured, as it is in the state which Ollivier designates *kirronese* or *coloration ictérique*. These malformations or morbid conditions, however, are of theoretical rather than of practical interest: and therefore they do not form fit subjects for further notice in an article like the present, which has solely a practical end in view.

B. PARTIAL DISEASES OF THE NERVOUS SYSTEM (*continued*).

3. DISEASES OF THE NERVES.

NEURITIS AND NEUROMA.
NEURALGIA.
LOCAL PARALYSIS.

LOCAL SPASMS.
TORTICOLLIS.
LOCAL ANÆSTHESIA.

NEURITIS AND NEUROMA.

By J. Warburton Begbie, M.D., F.R.C.P.E.

Morbid appearances, the results of inflammatory action, are occasionally met with in nerves. Such are the consequences usually of injury; the nerves have been divided by a sharp instrument; or if independent of wounds, they are in all probability connected with rheumatism or gout. There seems no reason to doubt that inflammatory action may likewise extend to nerves from the contiguous tissues.

In its general characters Neuritis resembles the inflammation of fibrous tissue. The fibrous investing sheath of nerves, or neurilemma, is indeed its usual seat; the appearances of inflammatory action being for the most part limited to it, and only seen in the form of red softening of the nervous tissue itself when the inflammation has been of an intense description.

A doubt as to the spontaneous occurrence of Neuritis has been entertained and expressed by several authorities. Boerhaave, for example, writes: "Nemo forte unquam vidit inflammationem in nervo; hæc vero si contingat, in sola *tunica vaginali* hæret."[1] Others, again, with even greater inaccuracy, have maintained the frequent existence of Neuritis.[2] Pathologically the inflammation of nerves may be acute or chronic; and these two conditions are described by Rokitansky as follows: The marks of the former (acute) are—(*a*) Injection and redness. The injection presents a linear arrangement, and the redness is partly caused by injection, and partly by small extravasations. (*b*) Looseness, succulence, and swelling of the nervous cord, due to infiltration of serum into the tissue of the neurilemma, and into the sheaths between the primitive nervous filaments. The nerve has lost its smooth, white, glistening appearance; its neurilemma is opaque, and has a rough and wrinkled look. (*c*) Exudation. This is generally a greyish or yellowish-red gelatinous product, which sooner or later becomes firm. It occupies the sheath and tissue of the neurilemma, and is likewise effused between the primitive filaments themselves. (*d*) The cellular tissue around the nervous cord always participates in these changes;

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[1] De Morbis Nervorum.

[2] See on this point Animadversiones de Neuritide: Praxeos Medicæ Universæ Præcepta, auctore Josepho Frank; Partis secundæ volumen primum, Sectio secunda, p. 131. Also Elements of General and Pathological Anatomy, by David Craigie, M.D., p. 379.

it becomes injected, reddened, and infiltrated with a serous or sero-fibrinous fluid. Not only the neighbouring cellular tissue, but the sheaths of the muscles, the fascia, the subcutaneous cellular tissue, and the general integuments, become involved.

Such a degree of inflammation as that now described may terminate in *resolution*, occurring quickly or slowly in different cases, or in *induration* of the nerve, and a permanent loss of its function in whole or in part. If the latter be the result, the nerve continues thickened, and more or less misshapen, forming a greyish cord, which is sometimes marked with black pigment and crossed by varicose vessels. The nerve filaments diminish in size and finally disappear, this result being in part due to the pressure to which they are exposed by the inflammatory product, and in part to their interrupted nutrition, for the vessels are obliterated by the inflammatory process. (*e*) In a more intense inflammation the primitive nervous filaments are destroyed. They are found in a state of red or greyish or yellowish-red softening, while the neurilemma is easily torn. (*f*) The fluid product of the inflammation may be purulent; and if so, the nerve appears highly discoloured, and infiltrated with purulent fluid tinged with blood. The neurilemma is then much altered, and readily gives way, while the nerve is converted into a yellowish-red, brownish-red, or chocolate-coloured pulp. The cellular tissue surrounding the nerve becomes infiltrated with yellow fibrinous exudation, and abscesses are formed in its course. (*g*) Ulcerative destruction of the nerve is the next step. But if the progress of inflammation be stayed before that point is reached, granulations appear, which become progressively changed into cicatrix tissue, as is observed in the stump of a nerve after amputation. Nerves, however, resist for a lengthened period the suppurative and sanious destruction which may be going on around them.

Chronic Inflammation is characterised by the varicose state of the vessels of the affected nerve, by products which become indurated, and gradually increase in quantity, and by a change of the nerve to a slate or lead-grey colour. Sometimes the products are not deposited uniformly throughout the nerve, and then nodular swellings are formed on it.[1] Romberg, when directing attention to the anatomical knowledge we possess of sciatica, speaks of Neuritis being found, but of its rare occurence.[2] The same writer, however, refers to the possible production of Neuritis, by the sciatic plexus being dragged and irritated by the head of the child in a difficult labour. Valleix and Beau have described inflammation of nerves more systematically than other authors. The latter has at considerable length, in his interesting memoir on the subject, directed attention to "Intercostal Neuritis."[3] Reference has been made to the occurrence of a rheumatic or gouty

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[1] A Manual of Pathological Anatomy, by Carl Rokitansky. Sydenham Society's Translation, vol. iii. p. 462.

[2] Lehrbuch der Nervenkrankheiten des Menschen: Neuralgie des Huftnerven.

[3] Valleix, Guide du Médecin Praticien, t. iv. p. 299; also Traité des Névralgies. Beau, Archives Générales de Médecine, 4^{e} série, t. xiii. 1847.

Neuritis. Dr. G. B. Wood considers it to be highly probable that in a large proportion of cases rheumatism lies at the foundation of the disease.[1] And Dr. Garrod, while admitting, according to the usually received notion, that the nervous affections occurring in connexion with gout are generally functional, believes them sometimes to be dependent on inflammatory action, which, he adds, appears, so far as can be ascertained, to have the character of true gouty inflammation.[2]

The most characteristic symptom of Neuritis is pain, not limited to the precise seat of the inflammation, but felt in the course of the nerve, and sometimes to its minutest branches. Besides its severity, the pain in Neuritis possesses other distinctive features: it is darting, and tingling, and there often accompanies it a feeling of numbness. The pain has been further described as intermittent, but is more probably remittent, being, as long as the disease continues, never entirely absent. Tenderness over the affected nerve invariably exists. It is possible that in some forms of local palsies (*see* Local Paralysis from Nerve Disease) the loss of power, partial or complete, as well as the existence of various morbid sensations, of which formication is one, and perhaps the most common, is due to disorganization or other permanent change in the trunk of a nerve, resulting from inflammatory action.

It seems to be generally admitted, that the nerve most liable to such change is the sciatic; but the various branches of the brachial plexus, and especially the ulnar nerves, likewise suffer; and so in all probability do at times the other nerves in both lower extremities and trunk.

That inflammation may also attack the nerves of special sense, as Dr. Wood has conjectured, seems not improbable, particularly the nerves of hearing and of sight. Most assuredly a true gouty inflammation, apparently commencing, in some cases, in the nerves themselves, not unfrequently either damages or entirely destroys one or other of the delicate organs connected with these most important functions.

In the treatment of Neuritis the probable alliance of the affection with some peculiar diathetic condition, the gouty or rheumatic, or possibly with the syphilitic cachexia, must not be lost sight of.

Local abstraction of blood, and the application of emollient and anodyne poultices, rest, low diet, and the use of laxatives, are the chief remedies in cases of the acute Neuritis. When the disease is chronic, the use of blisters, issues, and even the cautery, has been recommended. Internally, besides opium or other narcotic for the relief of pain, it will be prudent to give a fair trial in both the acute and chronic Neuritis to quinine, and colchicum, the iodide and the bromide of potassium.

NEUROMA (Tumour of Nerve).—Growths of various sizes and natures occurring in the course of nerves had been described before the term Neuroma came to be applied to such. Dr. Robert Smith, in his valuable and elaborate memoir, makes a brief reference to the early history of the subject;[1] and so likewise does Mr. William Wood, in his important papers entitled, "Observations on Painful Subcutaneous Tubercle," and "On Neuroma."[2] The famous English surgeon, William Cheselden, is specially mentioned, as having given the first accurate account of the nervous tubercle, which has become familiar chiefly through the writings of Mr. Wood. "Immediately under the skin, upon the shin bone, I have twice seen little tumours, less than a pea, round and exceeding hard, and so painful that both cases were judged to be cancerous: they were cured by extirpating the tumour. But what was more extraordinary was a tumour of this kind, under the skin of the buttock, small as a pin's head, yet so painful that the least touch was insupportable, and the skin for half an inch round was emaciated; this, too, I extirpated, with so much of the skin as was emaciated, and some fat. The patient, who before the operation could not endure to set his leg on the ground, nor turn in his bed without exquisite pain, grew immediately easy, walked to his bed without any complaint, and was soon cured." The same writer describes and figures the cystic neuroma. "A tumour formed in the centre of the cubital (ulnar) nerve, a little above the bend of the arm; it was of the cystic kind, but contained a transparent jelly; the filaments of the nerve were divided and ran over its surface. This tumour occasioned a great numbness in all the parts that nerve leads to, and excessive pain upon the least touch or motion. This operation (for the removal of the tumour) was done but a few weeks since; the pain is entirely ceased, the numbness a little increased, and the limbs as yet not wasted."[3]

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[1] A Treatise on the Practice of Medicine, vol. ii. p. 843.

[2] The Nature and Treatment of Gout and Rheumatic Gout, p. 517.

The term Neuroma, or rather *Neuromes,* was first employed by M. Odier of Geneva. "Enfin," writes Odier, "on peut donner le nom de Neuromes à ces tumeurs mobiles, circonscrites et profondes, qui sont produites par le gonflement accidentel d'un nerf, à l'extrémité duquel la compression de la tumeur fait éprouver des crampes très-pénibles."[4]

There have been various classifications of neuromatous tumours attempted by pathologists, such as local and general—that is, as affecting one nerve, or several nerves; and, again, those which are the direct consequence of a morbid process, and those resulting from an original vice of conformation. Dr. Smith, rejecting these divisions, has suggested, as sufficient for practical purposes, that Neuromata should be considered as of two kinds: 1st, of spontaneous origin, or *Idiopathic;* 2d, as the result of wounds or other injuries of the nerves, and therefore *Traumatic.*

Before offering a brief description of these varieties, it may be well to direct attention a little more fully to the *painful subcutaneous tubercle,* which we have the authority of Dr. Hughes Bennett and other pathologists for stating "must be referred to this class of tumours,"[1] that is, neuromatous fibrous tumours.

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[1] A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma. Dublin, 1849.

[2] Transactions of the Medico-Chirurgical Society of Edinburgh, vol. iii. pp. 317 and 367.

[3] The Anatomy of the Human Body, 12th edit., London, 1784, pp. 136 and 256.

[4] Manuel de Médecine pratique, ou Sommaire d'un Cours gratuit, donné en 1800, 1801, et 1804, aux Officiers de Santé du département du Léman, par Louis Odier. Paris, 1811, p. 362.

"Although," remarks Dr. Smith, "pathologists have hitherto failed to discover anything like nervous structure in these tumours, I still incline to the opinion that they are connected with the minute filaments and ultimate ramifications of the nerves. Upon any other supposition it is, I conceive, impossible to offer a rational explanation to account for the dreadful severity of the sufferings which they induce." Sir J. Paget, who has carefully examined the "painful subcutaneous tumours," describes them as being formed of "either fibro-cellular or fibrous tissue, in either a rudimental or a perfect state." Alluding to a case described by the late Professor Miller, in his "Principles of Surgery," and by Professor Bennett, the same pathologist admits that their structure may sometimes be fibro-cartilaginous.[2]

Of this affection the first detailed account was given by the late Mr. William Wood of Edinburgh. After the publication of Mr. Wood's earlier papers,[3] cases were recorded by different observers, and in 1829 an instructive *résumé* of the whole subject was laid by him before the Medico-Chirurgical Society of Edinburgh, and appeared, as already mentioned, in its "Transactions."

This disease consists in the formation of a small lump or tubercle seated in the subcutaneous cellular tissue, immediately under the integuments, which retain their natural appearance. The tubercle is met with in different parts of the body, but most frequently in the extremities. It is extremely small, pisiform in shape, of firm consistence, and apparently quite circumscribed.

The characteristic feature of the disease is the occurrence of violent pain coming on paroxysmally. The paroxysms vary in duration from ten minutes to upwards of two hours, their frequency as well as intensity appearing to increase in precise relation to the length of time the disease has existed. Some patients enjoy intervals of relief from pain for days or even weeks, while others have repeated attacks in the course of a single day. The paroxysms of pain frequently occur when the patient has fallen asleep. They are also apt to be excited by various external causes, such as pressure and blows; while in rarer instances mental disquietude and atmospheric changes have been their only apparent occasion.

Females are more frequently the subjects of this disease than males. Wood, referring to thirty-five cases collected by him, mentions that twenty-eight were females, five males; and in the account of two the sex was not stated. Of thirteen cases quoted by Descot, ten occurred in females, and three in males. Romberg has met with three instances, all in females.

The situation of the tubercle in the thirty-five cases referred to by

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[1] Clinical Lectures on the Principles and Practice of Medicine. 3d edit. p. 171.

[2] Lectures on Surgical Pathology, vol. ii. p. 123.

[3] The Edinburgh Medical and Surgical Journal, 1812. Two Articles, pp. 285 and 429.

Wood was as follows: in the lower extremities in twenty-two, in the upper extremities in eleven, in the chest in one, and in one in the scrotum. In only two of these cases was there more than one tubercle present.

This disease does not seem to be intimately connected with any particular period of life, as it has been noticed at all ages from thirteen to above seventy.

"It is a happy circumstance that this very painful affection is capable of being remedied by a very simple operation. The tubercle is easily removed by a single incision, and it is unnecessary to take away any portion of the integuments, or of the surrounding cellular tissue. No bad effect can follow the removal of the little body." (Wood.)

Although this subcutaneous tubercle has been considered as a variety of Neuroma, it must be held in remembrance that, its distinct connexion with branches of nervous trunks never having been determined, this is more a matter of inference than of demonstration. Ollivier and Rayer together carefully dissected the tumour in a case to which reference is made in his latest paper by Wood, and the result is thus expressed: "Extérieurement il était enveloppé de tissu cellulaire, dans lequel nous ne pûmes distinguer aucun filet nerveux, même à l'aide d'une forte loupe."[1] Paget remarks that the general opinion is against the supposition of the intimate connexion of these painful tumours with nerves. "Dupuytren," he writes, "says that he dissected several of these tumours with minute care, and never saw the slightest nervous filament adhering to their surface. I have sought them with as little success with the microscope. Of course I may have overlooked nerve-fibres that really existed. It is very hard to prove a negative in such cases; and cases of genuine Neuroma, *i.e.* of a fibrous tumour within the sheath of a nerve, do sometimes occur, which exactly imitate the cases of painful subcutaneous tumour."

We have now to consider the first of the two forms of Neuroma, as distinguished by Smith, and now generally recognised,—namely, the *Idiopathic Neuroma*. Tumours of this nature are of an oval or oblong form, their long axis corresponding with the direction of the nerve to which they are attached. They vary considerably in size. One figured in his work by Smith is as small as a grain of wheat, while another is as large as a good-sized melon. Between these two extremes every variety of size occurs. There may be only one, or several may be found on the same nerve; occasionally they are found existing simultaneously upon all the spinal nerves. "In number," says Rokitansky, "they vary from one until they are almost countless." A remarkable general disease is thus constituted, of which three cases have been observed in the Vienna Hospital. Neuromata are comparatively rare in the ganglionic system. But although occurring most frequently on the spinal nerves, Neuroma is not limited to them; the cerebral nerves, motor as well as sensory, particularly those most closely resembling the nerves of the cord, present at times the same tumours.

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[1] Traité théorétique et pratique des Maladies de la Peau, seconde édit., t. ii. p. 290. Paris, 1835.

In general, Neuromata are solid throughout their entire structure, but in some instances are of cystic formation, as in the case recorded by Cheselden, and already referred to. These tumours are of slow growth, but continue to undergo a steady increase in size, although many years may elapse before they attain such dimensions as to prove a source of serious inconvenience. They are moveable in the transverse direction, but not in the course of the nerve upon which they are seated. There may be a difficulty in distinguishing tumours which are merely contiguous to nerves from the true Neuroma, having its origin within the neurilemma. Wood has specially alluded to this difficulty in diagnosis, and Smith has pointed out that the non-nervous tumours, unlike Neuromata, are generally moveable in all directions, and, when drawn away from the nerve, cease to be painful on pressure.

Nerve tumours are described by Rokitansky as lying between the fasciculi of the nerves, and interwoven with their neurilemmatous sheath. Neuroma, the same pathologist observes, is never deposited in the centre of a nerve, but at its side, so that only a small part of its fasciculi is displaced; the displaced fasciculi are spread abroad and stretched over the tumour, while the greater mass of the nerve remains on the other side uninjured, and with its fibres in connexion with one another.

The solid neuromatous swellings are of a tough elastic consistence, of greyish or pale yellowish-red colour, and are invested with a distinct fibrous sheath. Dr. Hughes Bennett thus describes them:—"On being minutely examined, they are found to consist of fibrous texture more or less dense, the filaments often arranged in wavy bundles running parallel to each other, but occasionally assuming a looped form, or intercrossing with each other. I have also found them to contain groups of cells. Not unfrequently they are fibro-cartilaginous; sometimes with the cells closely aggregated together, at others widely scattered. In some of the neuromatous swellings described by Dr. Smith I found the fibrous tissue to present wavy bundles, among which a few granule and cartilage cells were scattered and shrivelled, apparently from the action of spirit."[1]

Neuromata seldom contract adhesion to the investing integuments, unless they have been subjected to continued pressure. Smith has never known them to suppurate, or to be removed by absorption. Pain has been generally considered to be a characteristic feature of neuromatous swellings. In this respect, however, there is infinite variety. When a single Neuroma exists, there is almost invariably much suffering. The pain, moreover, occurs suddenly and paroxysmally, darting along the nerve with the violence and instantaneousness of an electric shock. On the other hand, in those examples of Neuroma which are distinguished by the number of the tumours, it is not uncommon to find these occasioning little or no inconvenience to the patient.

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[1] Loc. cit. p. 171.

It is exceedingly difficult to determine with anything like exactness the real cause of the paroxysmal attacks and sudden aggravation of severe pain which occur in this as well as in many other forms of disease of the nervous system. Mental emotions and the ordinary atmospherical vicissitudes have been generally assigned as the occasion of these occurrences in Neuroma.

Paget has some very interesting observations on the cause of pain in Neuroma, as well as on the nearly entire absence of all suffering which has been noticed in some cases; and founding on the observations of Smith and others, including himself, this excellent writer is no doubt correct when he states "that we cannot assign the pain in these cases entirely to an altered mechanical condition of nerve-fibres in or near the tumour. We must admit, though it be a vague expression, that the pain is of the nature of that morbid state of nerve force which we call *neuralgic*. Of the exact nature of this neuralgic state indeed, we know nothing; but of its existence as a morbid state of nerve-force, or nervous action, we are aware in many cases in which we can as yet trace no organic change, and in many more in which the sensible organic change of the nerves is inadequate to the explanation of the pain felt through them." In short, Paget argues for the pain being functional, and not necessarily dependent at least on an organic disorder. If such a pain is found to be influenced by the remedies chiefly available for the relief of ordinary neuralgia—quinine, iron, arsenic, belladonna, stramonium, the bromide of potassium—this suggestion will receive corroboration.

We now know that such Neuromata as are the seat of severe pain and of continual irritation may give rise to attacks of the so-called sympathetic epilepsy. Instances of this nature are to be found in the writings of several authors, and it is sufficient here to refer to the well-known views of Brown-Séquard respecting the exciting causes of the epileptic convulsion, and of many other nervous affections.[1]

In the idiopathic form of Neuroma the pain is generally limited to the parts below the tumour; and the sign of the true Neuroma, signalized by Aransoohn, has been accepted by others—namely, that when the trunk of the nerve is compressed above the tumour the pain ceases, and then the Neuroma previously acutely sensitive can be touched without any uneasiness being caused. The remark already made as to the solid variety of Neuroma not being necessarily painful applies likewise to the fluid or cystic tumour.

Our knowledge of the determining causes of Neuroma cannot be said to have advanced since the period when the important treatise of

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[1] Researches on Epilepsy, p. 35; also Course of Lectures on the Physiology and Pathology of the Central Nervous System, p. 181. Article Neuroma, by same Author, in Holmes's System of Surgery, vol. iii. p. 896.

Dr. Smith first appeared, and we are still compelled to adopt his expression, "I feel it must be confessed that we know nothing with certainty regarding the causes of Neuroma."[1]

Neuromatous tumours have been frequently removed along with the corresponding portion of the nerve on which they were situated; and such operations, while entirely relieving the patients from suffering, have not been succeeded by any considerable loss of sensibility, or of the power of voluntary movement, in the parts supplied by even large nerves.

The sciatic nerve may be divided, as in a case of severe neuralgia of that nerve, by M. Malagodi, and a portion of it excised, without permanently destroying the functions of the limb.

The magnitude of the nervous trunk, which is the seat of the disease, will of course largely determine the period at which complete or partial restoration of the function in the limb is established. In some cases a few months, in others a year and upwards, have elapsed; but sooner or later, in all recorded instances, the banished sensibility and motor power have been regained.

The interference with the calorific function of the nervous system is strikingly exhibited in cases of operation for Neuroma. Mr. Adams and Dr. Smith have drawn attention to the diminution of temperature in the limb after the removal of the tumour, and with it a portion of nerve—a diminution readily noticed both by patients and operator, and which has lasted for a lengthened period, even after the restoration of the other functions.

It may then be concluded that when idiopathic Neuroma is seated in the hand, fore-arm, or upper arm (the positions in which it has most commonly been found), the operation of removal may be safely practised. It is possible that a similar plan might be adopted in the case of Neuroma in the lower extremity; but it is on record that amputation of the limb has been had recourse to by Chelius, in a case of nervous tumour occupying the popliteal space and stretching to nearly the centre of the back of the thigh. This was an illustration, and there are others which teach a similar lesson, of the disease having been permitted to attain a very large size—so large as to prevent any attempt being made for its simple removal.

Traumatic Neuroma.—Under this division are to be included tumours of nerves resulting from any form of mechanical injury, such as wounds, blows, pressure, or following amputation.

Traumatic Neuroma is almost invariably single. The tumour is the seat of intense pain, which, unlike the suffering in the idiopathic form of the disease, is not confined to the growth itself, or felt merely in the parts below it, but is frequently found extending along the nerve towards its origin. When Neuroma occurs as a consequence of a wound of nerve, it usually consists of a solid tumour, not invested by neurilemma, and destitute of any distinct capsule.[1] It is most likely to form, when the nervous cord has been cut, but not entirely divided; and cases of this nature are even more than ordinarily painful.

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[1] Loc. cit. p. 5.

The following case is published by Mr. Wood in his "Memoir on Neuroma;" it occurred in the practice of Mr. Syme:—

"James Muir, aged 43. 30*th June*, 1828.—On the inner side of the left knee, about a hand-breadth above the joint, there is a narrow depressed cicatrix, two inches long. Between this cicatrix and the sartorius there is a small tumour, about the size of an almond, and of very firm consistence. When the limb is extended, this tumour can hardly be perceived, being then overlapped by the sartorius; but when the knee is bent, it can be felt very distinctly. It is most moveable in a lateral direction, but seems pretty firmly connected to the subjacent parts by condensed cellular substance.

"The patient states that the tumour is always painful when pressed, but is more so at one time than another. The pain is not confined to the part, but shoots all over the knee, and sometimes extends from the groin to the toes. He observes that the pain is more severe during cold and damp weather. It frequently, for days together, prevents him from walking, or even resting on the limb. His story is, that when a boy, about eleven years old, he strained his knee by jumping into a saw-pit, which led to the formation of a large abscess that opened on both sides of the knee, namely, at the part where the cicatrix above mentioned still remains, and exactly opposite, where also there is a similar cicatrix. Several small bits of bone were discharged, and at the end of two years he got quite well. For the following twenty-seven years he led an active life; ten of them were spent in a militia regiment. About eight years ago he strained his knee while walking in his garden, and thereafter became subject to flying pains about the joints. These pains induced him to rub the knee frequently; and in doing so, about two years ago, he noticed the tumour. It was then the size of a pea, and has gradually enlarged. The disagreeable symptoms also have become greatly aggravated; and, as he refers them all to the tumour, he is desirous of having it removed.

"12*th July*.—Mr. Wood (continues Mr. Syme), who was kind enough to examine the patient, having agreed with me that the tumour was seated on or in the nervus saphenus, and that it ought to be removed, I performed the operation, with his assistance, on the 1st of July.

"The tumour being divided showed a firm fibrous capsule, containing a soft brownish-white pulpy matter. The nerve was traced into the tumour, but not through it. The patient made a good recovery, and remains free from his complaint."[2]

The foregoing case illustrates the proper treatment of Traumatic Neuroma, which is to excise the tumour with the corresponding portion of nerve, in every case when its situation will permit of this being done.[1]

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[1] Smith, loc. cit. p. 20.

[2] Loc. cit. p. 426.

The last form of Neuroma which requires any separate consideration is that succeeding to amputations. Smith remarks in regard to such, that "their existence is so constant that we may, perhaps, consider them as representing the normal condition of the ends of the nerves in stumps." Generally they cause no uneasiness whatever: but, on the other hand, they have occasionally been the source of severe neuralgia, occurring in paroxysms of great length.

The Neuroma of stumps varies in size, being in some instances not larger than a garden-pea, in others as large as a grape, or even plum. Such Neuromata are generally of an oval or oblong form, of greyish-white colour, and of a firm dense texture.

The situation of the Neuroma in the stump is not always the same; it may be several inches above the surface of the latter, and be connected with the cicatrix by means only of a fibrous cord, itself destitute of any nervous structure.

It is the opinion of some pathologists, that the Neuromata succeeding amputation are produced by the pressure which is exerted upon the surface of the stump. An objection fatal to this explanation, however, has been advanced—namely, that in many stumps which have never been subjected to pressure these little tumours are found.

Dr. Smith believes their formation to be for the protection of the extremity of the nerve.

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[1] Smith, p. 22.

NEURALGIA.

By Francis Edmund Anstie, M.D., F.R.C.P.

Definition.—A disease of the nervous system manifesting itself by pains, nearly always unilateral, which appear to follow the course of particular sensory nerves. The pains are usually sudden in their commencement, and of a darting, stabbing, boring, or burning character; they are at first unattended with any local change which can be recognised, or by any constitutional pyrexia. They are always markedly intermittent; sometimes regularly and sometimes irregularly so. The periods of intermission are distinguished by complete freedom from acute suffering, and in recent cases the patient appears quite well at these times. In old standing cases, however, persistent tenderness and other signs of local mischief are apt to be developed in the tissues which surround the distribution of the nerves which are the seat of the acute pains. Severe attacks of Neuralgia are usually complicated with secondary affections of other nerves which are intimately connected with that which is the original seat of pain; and in this way congestion of blood-vessels, hypersecretion, or arrested secretion from glands, inflammation and ulceration of tissues, &c., are sometimes brought about.

Synonyms.—The word Neuralgia has a generally recognised force, and there is no equivalent to it (except foreign variations in mere terminology) which represents the whole group of disorders to which it applies, though there are numerous phrases for particular forms of the disorder.

Clinical History and Symptoms.—These vary so greatly in different cases of Neuralgia that it will be necessary to discuss the greater part of this subject under the headings of the special varieties of the disease. There are certain features, however, which are observed in all true Neuralgias.

In the first place, it is universally the case that the existing condition of the patient at the time of the first onset of the disease is one of debility, either general or special. I make this statement with great confidence, notwithstanding the contrary assertion advanced by so high an authority as Valleix, whose able[1] treatise really laid the foundation for all our accurate knowledge of the Neuralgias. In the first place, it is certainly the case that the larger half of the total number of patients coming under my care with various forms of Neuralgia are either decidedly anæmic or have recently undergone some exhausting illness or fatigue: and the reason why Valleix did not find so many cases of this type among his neuralgic patients appears certainly to be, that he limited the neuralgic class of diseases by an artificial definition, which we shall have to reject as untenable. On the other hand, although a considerable number of neuralgic patients are so far healthy in appearance, that they have a fairly ruddy complexion and a good amount of muscular strength, it is impossible to admit that these facts disprove the existence of debility, either structural or functional, *in the nervous system,* for the commonest experience teaches that such debility does frequently co-exist with a great robustness and development of the apparatus of vegetation and the lowest forms of animal function. And it will invariably be found, on carefully examining these apparently robust neuralgic patients, that the nervous system has given warnings of its weakness: thus, the patient who, after an exhausting confinement, attended with great loss of blood, is attacked with obstinate *clavus hystericus,* will inform us that whenever in earlier life she had suffered from headache, the pain was always chiefly, if not altogether, confined to the nerves which are now the seat of decided Neuralgia. In a large number of cases I have also found that the attack of acute pain was immediately ushered in by a remarkably *anæsthetic* condition of the parts about to become painful; and a slighter degree of blunted sensation may often be observed in the intervals between the earlier attacks in cases of Neuralgia. In short, I have never seen a case of neuralgic pain in which there were not marked evidences of nervous debility, either local or general.

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[1] Traité des Névralgies. Paris, 1841.

Another circumstance is common to all Neuralgias of superficial nerves; and as a large majority of neuralgic affections are superficial in situation, this is, for practical purposes, a general characteristic of the disease. I refer to the formation of *tender spots* at various points where the affected nerves pass from a deeper to a more superficial level, and particularly where they emerge from bony canals, or pierce fibrous fasciæ. So general is this characteristic of inveterate cases, that Valleix founded his diagnosis of the genuine Neuralgias on the presence of these painful points, in which assumption I think there can be little doubt, that he committed an error.[1]

The third general characteristic of neuralgic affections is, that the pain is intermittent, or at the least remittent, in every stage of the disease.

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[1] Trousseau insists with much energy that a still more important "point douloureux" is constantly present in Neuralgia, viz. over the spinous processes of one or more vertebræ, corresponding to the origin of the painful nerve. It is true (as the Brothers Griffin had long before pointed out), that there is *tenderness* in this situation. But this "point apophysaire" is not always, nor frequently, the seat of *spontaneous pain.*

The fourth general characteristic is, that fatigue and every other temporary depressing influence directly predisposes to an attack of acute pain, and aggravates it when already existent.

Varieties.—It is possible to classify the Neuralgias upon either of two systems: first (A), according to the constitutional condition of the patient; and, secondly (B), according to the situation of the affected nerves. It will be necessary to follow both these lines of classification, avoiding repetition as much as possible.

(A) In considering the influence of constitutional states upon the typical development of Neuralgia, it will be convenient to commence with (I.) the group of cases in which the general state of the organism exerts the least amount of effect. This is the case where the pain is the result of direct injury to a nerve-trunk, whether by external violence, by the mechanical pressure of a tumour, or by the involvement of a nerve in inflammatory or ulcerative processes, spreading to it from neighbouring tissues. As regards the development of symptoms, the important matters are, that the pain in these cases commences comparatively gradually, that the intermissions are usually much less complete, and that the pain is far less amenable to relief from remedies than in other varieties of Neuralgia. The little that can be said about the form which is dependent upon progressively increasing pressure, or involvement of a nerve in malignant ulcerations, caries of bones, or teeth, &c., falls under the heads of Diagnosis or Treatment, and need not detain us here. The clinical history of Neuralgia from external violence, however, requires separate discussion.

1. Neuralgia from external violence may be produced by a shock (as of a fall, a railway collision, &c.), which gives a jar to the central nervous system, or by severe mental emotion, operating upon the same part of the organism. Upon either of these circumstances the development of the affection seldom occurs at once, but ensues after a variable interval, during which the patient exhibits symptoms of a general depression, with loss of appetite and strength. Sometimes vomiting, and even, in other instances, actual paralysis of a partial and temporary kind occur. When once developed, the neuralgic attacks are undistinguishable from those which occur from causes internal to the organism. The affection is usually very obstinate. In a large number of cases the nerve or nerves affected have previously shown signs of weakness, by a tendency to painful affection in depressed states of the organism. In the greater number of instances, as far as my experience goes, it is the fifth cranial nerve which becomes neuralgic from the effects of central shock. Illustrative cases will be given in the sections on local classification.

2. Neuralgia from direct violence to superficial nerves is produced either by cutting or, more rarely, by bruising wounds.

Cutting wounds may divide a nerve-trunk, (α) partially, or (β) completely.

(α) When a nerve-trunk is partially cut through, neuralgic pain commonly occurs, if at all, immediately on the receipt of the injury. One such example only has come under my own care, but many others are recorded.[1] In this case the ulnar nerve was partly cut through with a tolerably sharp bread-knife, at a point not far above the wrist; partial anæsthesia of the little and ring fingers was induced, but at the same time violent neuralgic pains in the little finger came on, in fits recurring several times daily, and lasting for about half a minute. Treatment was of little apparent effect in promoting cure, though opiates gave temporary relief, as did the local use of chloroform. The attacks recurred for more than a month, long after the original wound had healed soundly; and for a long time after this pressure on the cicatrix would reproduce the attacks. A slight amount of anæsthesia still remained when I last saw the patient, more than a year after the injury.

(β) Complete severance of a nerve-trunk is a sufficiently common accident, far more common than is the production of Neuralgia from such a cause; indeed so marked is this disproportion between the injury and the special result, that I have been led to the conclusion that a necessary factor in the chain of morbid events must be the existence of some antecedent peculiarity of organization in the central origin of the injured nerve. This opinion is rendered more probable by the fact that the consecutive Neuralgia is not unfrequently situated not in the injured nerve itself, but in some other nerve with which it has intimate central connexions. Two such examples are recorded in my Lettsomian Lectures,[1] in which the ulnar nerve, and one in which the cervico-occipital, respectively, were completely divided: in all three instances the Neuralgia was developed in the branches of the trigeminus. In all the cases which have come under my notice the Neuralgia, whether direct or reflex, set in at a particular period, viz. after complete cicatrization of the wound, and while the functions of the branches on the peripheral side were partly, but not completely, restored. The same obstinacy and rebelliousness to treatment was noticed as in other instances of Neuralgia from injury.

A few words must be given, before quitting the subject of Neuralgia from wounds of nerves, to the cases in which a foreign body lodges, with more or less laceration, in the substance of a nerve trunk. I have never seen such a case; but many instances are recorded in which most violent and painful Neuralgia has been set up in this way. Not unfrequently the irritation produces no noticeable effect on the nerve actually pressed upon, but sets up Neuralgia in a nerve so distant that no connexion is suspected between the neuralgic pain and the original accident. The removal of a small piece of glass, or such other irritating body from the cicatrix of an old wound, has in several recorded instances put an end to neuralgic pains in quite another situation, for which all manner of remedies had long been tried. Sometimes the neuralgic pain has been accompanied by tissue degeneration of an alarming character, and these have likewise ceased at once upon the removal of the peccant body which had been the unsuspected source of the evil.

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[1] *Vide* Lancet, 1866.

Neuralgias which result from some local injuries of so peculiar a character as *gun-shot wounds* scarcely fall properly within the province of this article. The reader who desires to know all that can be said with regard to this particular class of affections is recommended to study the able and carefully compiled "Report" of Messrs. Mitchell, Morehouse, and Keen.[1]

The case of Neuralgia from injury, pressure, and local disease of nerves has been mentioned first, because this form of the disease is less influenced than others by general constitutional states. But it is an erroneous opinion, however common, that the general condition of the body is here without any influence on the development of the nerve-pain. It has been forcibly urged, by Dr. Brinton and Dr. Handfield Jones more especially, that a condition of general bodily vigour mitigates, and that constitutional debility decidedly aggravates, these forms of Neuralgia; and my own experience gives most practical proof of the justice of this argument.

(II.) *Neuralgias of intra-nervous origin.*—As regards the constitutional conditions with which the several varieties of Neuralgia that arise independently of external violence, or disease of extra-nervous tissues, are respectively allied, the following preliminary subdivision may be made:—

1. Neuralgias of malarious origin.
2. Neuralgias of the period of bodily development.
3. Neuralgias of the middle period of life.
4. Neuralgias of the period of bodily decay.
5. Neuralgias associated with anæmia and mal-nutrition.

1. *Neuralgias of malarious origin* were formerly far more prevalent than they are at present, within the sphere of the English practitioner of medicine; with the general decline of malarial fevers, consequent on improved drainage and cultivation of lands, they have become constantly more scarce. In former times, on the contrary, they were so common, that they forced themselves on the notice of every physician. The term "brow-ague," to this day applied by many medical men to every variety of supra-orbital Neuralgia, is a relic of the older experience on this point; as is also the very common mistake of expecting all neuralgic affections to present a distinctly rhythmic recurrence of symptoms.

My own experience of malarial Neuralgia has been very limited, and I may as well say all that I know of its symptoms at once. In fact, though the out-patient practice of the Chelsea Dispensary and Westminster Hospital has afforded me a considerable number of examples of ague in past years, I have only seen two undoubted and one doubtful case of malarial Neuralgia, in all of which the fifth nerve was affected.

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[1] Report on Gunshot Injuries to Nerves, observed in the late American War. Philadelphia, 1864.

The periodicity in one of the genuine cases was regular tertian; in the other regular quotidian. An algide condition always ushered in the attacks; but this was gradually exchanged, as the pain continued, for a condition in which the pulse was rapid, soft, and bounding, and the strength was further depressed. In both of these cases there were unilateral flushing of the face, and congestion of the conjunctiva, to a slight degree, during the attack of pain. The pain became duller and more diffused contemporaneously with the lowering of arterial pressure (as estimated by Marey's Sphygmograph); and after the disappearance of active pain, moderate tenderness over a considerable tract around the course of painful nerves remained for some time. But there was no distinct development of the *painful points* of Valleix (to be hereafter described), a circumstance which I attribute to the rapid cure of the complaint, in each instance, by quinine.

2. *Neuralgias of the period of bodily development.*—By the "period of bodily development" is here understood the whole time from birth up to the twenty-fifth year, or thereabouts. This is the period during which the organs of vegetative and of the lower animal life are consolidating. The central nervous system is more slow in reaching its fullest development, and the brain more especially is many years later in acquiring its maximum of organic consistency and functional power.

That portion of the period of bodily development which is antecedent to puberty is but little obnoxious to neuralgic affections. From the moment when puberty arrives, however, all is changed. In the stir and tumult which pervades the organism, and especially in the enormous diversion of its nutritive and formative nisus to the evolution of the generative organs and the correlative sexual instincts, the delicate apparatus of the nervous system is apt to be overwhelmed, as well as left behind, in the race of development. Under these circumstances the tendency to neuralgic affections rapidly increases. It will, however, be seen later that there is a great preponderance of particular varieties of the disease among the cases occurring during this period.

3. *Neuralgias of the middle period of life.*—By this period is meant the time included between the twenty-fifth and about the fortieth or the forty-fifth year. It is the time of life during which the individual is subjected to the most serious pressure from external influences. The men, if poor, are engaged in the absorbing struggle for existence and for the maintenance of their families; or, if rich and idle, are immersed in dissipation, or haunted by the mental disgust which is generated by *ennui.* The women are going through the exhausting process of childbearing, and supporting the numerous cares of a poor household in some cases, or are devoured with anxiety for a certain position in fashionable society for themselves and their children, or again they are idle and heart-weary, or condemned to an unnatural celibacy. Very often they are both idle and anxious.

It must not be supposed that there is a sharp line of demarcation between this period and the last: nevertheless it will be seen, when we come to discuss the local varieties of Neuralgia, that there are certain broad differences in the general tendencies of the two epochs. It must be noted that particular Neuralgias, which are first manifested in the development period, frequently recur, under special provocation, in the period of middle life.

4. *Neuralgias of declining bodily vigour.*—The period here referred to is that which commences with the first indications of distinct physical decay, of which the earliest that we can recognise (in persons who are not cut off by special diseases) is perhaps the tendency to atheromatous change in the arteries. The earliest development of this system varies very considerably in date; but whenever it occurs it is a plain warning that a new set of vital conditions has arisen; and especially notable is its connexion with the characters of the neuralgic affections which take their rise after its commencement. The period of declining life is pre-eminently the time for *severe and intractable* Neuralgias. Very few patients indeed are ever permanently cured, who are first attacked with Neuralgia after they have entered upon what may be called the "degenerative" period of existence.

Perhaps a separate heading should be reserved for those Neuralgias which are the heralds of locomotor ataxy. But they seem naturally to fall under the present class, although the nervous degeneration which produces them is chiefly in the direction of sclerosis. The character of these pains is fully described in the article on Locomotor Ataxy.

5. *Neuralgias which are immediately excited by anœmia or mal-nutrition.*—Of the neuralgic affections which can be ranked within this group, the sole characteristic worthy of note here is the circumstances in which they arise. It would seem that conditions of anæmia and mal-nutrition simply aggravate the tendencies of existing weak portions of the nervous system to be affected with pain; just as they notoriously do aggravate lurking tendencies to convulsion and spasms.

(B.) We come now to the consideration of local varieties of Neuralgia. The primary subdivision of these may be made as follows:—

(I.) Superficial Neuralgias. (II.) Visceral Neuralgias. The superficial Neuralgias may be subdivided thus:

(*a*) Neuralgia of the fifth (trifacial or trigeminal) nerve.
(*b*) Cervico-occipital Neuralgia.
(*c*) Cervico-brachial Neuralgia.
(*d*) Intercostal Neuralgia.
(*e*) Lumbo-abdominal Neuralgia.
(*f*) Crural Neuralgia.
(*g*) Sciatic Neuralgia.

This classification is taken from Valleix, and appears to me substantially correct.

(*a*) The most important group of Neuralgias are those of the fifth cranial nerve.

Neuralgia of the fifth nerve always exhibits itself with especial violence in certain foci, which Valleix was the first to define with accuracy. These foci are always in points where the nerve becomes more superficial, either in turning out of a bony canal, or in penetrating fasciæ. In the ophthalmic division of the nerve the following possible foci are noticeable: (1) the *supra-orbital*, at the notch of that name, or a little higher in the course of the frontal nerve; (2) the *palpebral*, in the upper eyelid; (3) the *nasal*, at the point of emergence of the long nasal branch, at the junction of the nasal bone with the cartilage; (4) the *ocular*, a somewhat indefinite focus within the *globe* of the eye; (5) the *trochlear*, at the inner angle of the orbit.

In the superior maxillary division the following foci may be found: (1) the *infra-orbital*, corresponding to the emergence of the nerve of that name from its bony canal; (2) the *malar*, on the most prominent portion of the malar bone; (3) a vague and indeterminate focus, somewhere on the line of the gums of the upper jaw; (4) the superior *labial* point, a vague and not often an important focus; (5) the *palatine* point, rarely observed, but in some recorded cases the seat of intolerable pain.

In the inferior maxillary division the foci are:—(1) the *temporal*, a point on the auriculo-temporal branch, a little in front of the ear; (2) the *inferior dental* point, opposite the emergence of the nerve of that name; (3) the *lingual* point (not a common one) on the side of the tongue; (4) an *inferior labial* point, one rarely met with.

Besides these foci in relation with distinct branches of the trigeminus, there is one of especial frequency, which corresponds to the *inosculation of various branches*. This is the *parietal* point, situated a little above the parietal eminence. It is small in size; the point of the little finger would cover it. It is the commonest foci of all.

Neuralgia of the fifth nerve may attack any one, or all three of the divisions; the latter event is comparatively rare.[1] The most common is the case of its limitation to the ophthalmic division, and incomparably the most frequent foci of the pain are the *supra-orbital* and *parietal* points.

The most common of all the varieties of trigeminal Neuralgia is Migraine, or sick-headache. This is an affection which is entirely independent of digestive disturbances, in its primary origin, though it may be aggravated by their occurrence. It almost always first attacks individuals at some time during the period of bodily development. Under the influences proper to this vital epoch, and often of a further debility induced by precocious straining of the mental powers, the patient begins to suffer headache after any unusual fatigue or excitement, sometimes without any distinct cause of this kind. The unilateral character of this pain is not always detected at first; but as the attacks increase in frequency and severity, it becomes obvious that the pain is limited to the supra-orbital, and sometimes to the ocular branches of the ophthalmic division of the fifth nerve of one side. In very rare cases, however, as in all forms of Neuralgia, the nerves of both sides may be affected. If the pain lasts for any considerable time, nausea, and at length vomiting, are induced. This is followed at the moment by the increase of the severity of the pain; but from this point the violence of the affection begins to subside, and the patient usually falls asleep. The history of the attacks negatives the idea that the vomiting is ordinarily remedial. This symptom merely indicates the lowest point of nervous depression; but it may happen that a quantity of food which has been incautiously taken, lying, as it does, undigested in the stomach, may of itself greatly aggravate the Neuralgia, by irritation transmitted to the medulla oblongata. In such a case vomiting may directly relieve the nerve-pain. When the patient awakes from sleep, the active pain is gone. But it is a common occurrence, indeed it always happens when the Neuralgia has lasted a certain length of time, that a *tender* condition of the superficial parts remains for some hours, perhaps for a day or two. This tenderness is usually somewhat diffused, and not limited with accuracy to the foci of greatest pains during the attacks.

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[1] It is with much diffidence that I make this statement, as it is opposed to the opinion of Valleix. But my own experience is very positive on the matter; and, besides, it appears to me that Valleix's definition of Neuralgia, which I cannot accept as sufficiently expansive, accounts for his views.

Sick-headache is not uncommonly ushered in by sighing, yawning, and *shuddering*—symptoms which remind us of the prodromata of some graver neuroses, to which it is probably related by hereditary descent.

Another variety of trigeminal Neuralgia which infests the period of bodily development is that known as *clavus hystericus; clavus* from the fact that the pain is at once severe, and limited to one or two small definite points, as if a nail or nails had been driven into the skull. These points correspond either to the *supra-orbital* or the *parietal;* sometimes both these are the seat of the pain. But for the greater limitation of the painful area in clavus, that affection would scarcely differ from migraine, for the former is also accompanied, when the pain continues long enough, with nausea and vomiting. The adjective *hystericus* is an improper and inadequate definition of the circumstances under which clavus arises. The truth is that the subjects of it are usually females who are passing through the trying period of bodily development; but there is no evidence to show that uterine disorders give any special bias towards this complaint. Both migraine and clavus are often met with in persons who have long passed the period of bodily development. But their first attacks have nearly always occurred during that period of life.

The adult or middle period of life is not, according to my experience, fruitful in *first attacks* of trigeminal Neuralgia. But when the neuralgic tendency has once been set up, there are many circumstances of middle-adult life which tend to recall it. Over-exertion of the mind is one of the most frequent; more especially when this is accompanied by anxiety and worry; indeed the latter is a more powerful cause than the former. In women, the exhaustion of hæmorrhage at parturition, or of menorrhagia, and also the depression produced by over-lactation, are frequent causes of the recurrence of a migraine or a clavus to which they had been subject when young. The middle period of life is also most obnoxious, on the whole, to severe mental shocks, and also to severe bodily accident, of a kind to produce damage to the central nervous system. Special mention ought to be made in the case of women, of the disturbing influences of the great series of changes which close the middle period of their life,—viz. the involution of the sexual organs. This is doubtless a very frequent cause of the resuscitation of a tendency to facial Neuralgia which had lain dormant, perhaps, for many years.

It is, however, the final or degenerative period of life which produces the most formidable varieties of facial Neuralgia. Neuralgias of the fifth which have previously attacked an individual, may recur at this time of life without any special character except a certain increase of severity and obstinacy. But trigeminal Neuralgias which now occur for the first time are usually intensely severe and utterly incurable. These cases correspond with the affection named by Trousseau "tic épileptiforme," and it is of them, doubtless, that Romberg is speaking, when he says that the true Neuralgias of the fifth rarely occur before the fortieth year of life. These affections are distinguished by the intense severity of the pain, the lightning-like suddenness of its onset, and the almost total impossibility of effecting more than the most temporary improvement in the symptoms. But they are also distinguished by another circumstance which too often escapes attention; namely, they are almost invariably connected with a family taint of insanity, and very often with strong melancholy and suicidal tendencies in the patient himself, which do not depend on, nor are commensurate in their development with, the intensity of the pain which he suffers. They are further remarkable for the frequency with which they are attended with two special complications—viz. muscular spasms, and the formation of exquisitely tender points, the least pressure on which is enough to cause the most violent agony. Often, a mere breath of wind impinging on them will produce a like effect. The history of these cases is most wretched: the unfortunate patient may survive for years before he completely succumbs to exhaustion; yet every hour of his life is a misery. The act of masticating usually causes intolerable darts of agony, and nutrition is often obliged to be kept up by liquids. If mere broth and slop diet be adhered to, there is probably under-nutrition, which aggravates the Neuralgia. And if, as often happens, the patient flies to drink as a relief, that again hastens the degeneration of the nervous centres, and renders the case more hopeless of cure than ever.

(*b*) *Cervico-occipital Neuralgia.*—As Valleix has remarked, there are several nerves (in fact the posterior branches of all the first four spinal pairs) which are more or less capable of being the seat of this affection. But amongst them all there is none comparable to the great occipital, which arises from the second spinal pair, for the frequency and importance of its neuralgic affections. This nerve sends branches to the whole occipital and the posterior parietal region. On the other hand, the second and third spinal nerves help to make up the superficial cervical branch of the cervical plexus, which is distributed to the triangle between the jaw, the median lines of the neck, and the edge of the sterno-mastoid, and those to the lower part of the cheek. Then there is the auricular branch, which starts from the same two pairs, and supplies the face, the parotid region, and the back of the external ear. Then, the small occipital, distributed to the ear and to the occiput. And finally there are the superficial descending branches of the plexus. These, altogether, are the nerves which, at various points, where they become more superficial, form the foci of cervico-occipital Neuralgia.

The most typical example of this form of Neuralgia which has fallen under my own notice, occurred (after exposure to cold wind) in a lady about sixty years of age, who had all her life been subject to neuralgic headache, approaching the type of migraine, and who came of a family in which insanity, apoplexy, and other grave neuroses had been frequent. The pain centred very decidedly in a focus corresponding to the occipital triangle of the neck. It occurred at irregular intervals, and in very severe paroxysms, and was entirely unaffected by any remedies, till *blistering* was tried, when it yielded at once. About twelve months later this patient suffered a severe hemiplegic attack of paralysis.

The tendency, however, of cervico-occipital Neuralgias, is certainly to spread towards the lower portions of the face, as observed by Valleix; in this case they become, sometimes, undistinguishable from Neuralgias of the third branch of the fifth. In the early stages of the disease, if the physician had been lucky enough to witness them, the true place of origin of the malady would have been easily discernible; at a later date it requires great care, and a very strict interrogation of the patient, to discover the true history of the disease.

Experience is too limited, if I am to judge by my own and that of the standard authors, to allow us to say anything of the conditions, as to age and general nutrition of the organism, which specially favour cervico-occipital Neuralgia. Apparently, however, there is good reason for thinking that the immediately exciting cause of it is most frequently *external cold.* But I am inclined to think also that it is seldom a primary Neuralgia, but occurs usually in subjects who have already experienced other forms.

(*c*) *Cervico-brachial Neuralgia.*—This class includes all the Neuralgias which occur in nerves originating from the brachial plexus, as from the posterior branches of the four lower cervical nerves. The most important characteristic of the Neuralgias of the upper extremity is the frequency, indeed almost constancy, with which they invade simultaneously or successively several of the nerves which are derived from the lower cervical pairs. The neuralgic affections of the small posterior branches (distributed to the skin of the lower and back part of the neck) are comparatively of slight importance. But the "solidarité," which Valleix so well remarked, between the various branches of the brachial plexus, causes the Neuralgias of the shoulder, the arm, fore-arm, and hand to be extremely troublesome and severe, owing to the numerous foci of pain which usually exist. Perhaps Valleix's description of these foci is somewhat fanciful and over-minute; but the following among those which he mentions I have repeatedly identified: (1) an *axillary* point, corresponding to the brachial plexus itself; (2) a *scapular* point, corresponding to the inferior angle of the scapula;—(it is difficult to identify the peccant nerve here: the one to which it apparently corresponds, and to which Valleix refers it, is the sub-scapular; but we are accustomed to think of this as a motor nerve. Still it is certain that pressure on a painful point existing here will often cause acute pain in the nerves of the arm and fore-arm:)—(3) a *shoulder* point, which corresponds to the emergence, through the deltoid muscle, of the superficial filets of the circumflex; (4) a *median-cephalic* point, at the bend of the elbow, where a branch of the musculo-cutaneous nerve lies immediately behind the median cephalic vein; (5) an *external humeral* point, about three inches above the elbow, on the outer side, corresponding to the emergence of the cutaneous branches which the musculo-spiral gives off as it leaves the groove in the humerus; (6) a *superior ulnar* point, corresponding to the course of the ulnar nerve, between the olecranon and the epi-trochlea; (7) an *inferior ulnar* point, where the nerve passes in front of the annular ligament of the wrist; (8) a *radial* point, making the place where the radial nerve becomes superficial at the lower and external aspect of the fore-arm. Beside these foci, there are sometimes, but more rarely, painful points developed by the side of the lower cervical vertebræ, corresponding to the posterior branches of the lower cervical pairs.

The most common seat of brachial Neuralgia in my experience has been the ulnar nerve; the superior and inferior points above mentioned being the foci of great intensity; an axillary point has also been developed in one or two instances which I have seen. Rarely, however, does the Neuralgia remain limited to the ulnar nerve; in the majority of cases it soon spreads to other nerves which emanate from the plexus. A very common seat of Neuralgia is also the shoulder, the affected nerves being the cutaneous branches of the circumflex. I am inclined to think, also, that affections of the musculo-spiral and of the radial near the wrist are rather common, and have found them extremely obstinate and difficult to deal with. One case has recently been under my care in which the foci of greatest intensity of pain were an external humeral, and a radial point; but beside this there was an exquisitely painful scapular point. In another instance, the pain commenced in an external humeral and a radial focus; but subsequently the shoulder branches of the circumflex became involved. A most plentiful crop of herpes was an intercurrent phenomenon in this case.

Median cephalic Neuralgia is an affection which used to be comparatively common in the days when phlebotomy was in fashion, the nerve being occasionally wounded in the operation. I have only seen it in connexion with this cause; that is to say, as a well-marked affection. One such instance has been under my care. But a slight degree of it is not uncommon, as a secondary symptom in Neuralgia affecting other nerves. The traumatic form is excessively obstinate.

In the Neuralgias of the arm we begin to recognise the etiological characteristic which distinguishes most of the neuralgic affections of limbs; namely, the frequency with which they are aggravated, and especially with which they are kept up and revived, when apparently dying out, by *muscular movements.* In the case above referred to, of Neuralgia of the sub-scapular, musculo-spiral (cutaneous branches), and radial, the act of playing on the piano for half an hour immediately revived the pains in fullest force, when convalescence had apparently been almost established.

The liability of particular nerves in the upper extremity to Neuralgia, from *external injuries*, requires a few words. The nerve which is probably most exposed to this is the ulnar. Blows on what is vulgarly called the funny bone are not uncommon exciting causes of the affection in predisposed persons: and cutting wounds of the ulnar a little above the wrist are rather frequent causes. The deltoid branches of the circumflex, and the humeral cutaneous branches of the musculo-spiral, are much exposed to injury. The radial nerve near the wrist is very much exposed both to bruises and to cutting wounds. So far as I know it is only when a nerve-trunk of some size is injured that Neuralgia is a probable result. Wounds of the small nervous branches in the fingers, for instance, are very seldom followed by Neuralgia. I have no statistics to guide me as to the effect of long-continued *irritation* applied to one of those small peripheral branches; but it is probable that that might be more capable of inducing Neuralgia. As far as my own experience goes, however, it would appear that a more common result is *convulsion* of some kind, from reflex irritation of the cord.

(*d*) *Dorso-Intercostal Neuralgia.*—This form of Neuralgia has of late years assumed a position of much interest, in consequence chiefly of its rather frequent association with unilateral herpes, a circumstance which has considerably helped to elucidate the pathology of the latter disease.

This disease is surrounded with considerable diagnostic difficulties. Some of these will be discussed under the head of Diagnosis in part; but a few words must be given to them here. The disorder with which it is especially liable to be confounded is that for which Dr. Inman invented the term Myalgia, and which is represented in different localities by the affections called in old-fashioned phrase pleurodynia, lumbago, and (more generally) by the very inaccurate term muscular rheumatism (there being no reliable evidence whatever to connect it specially with the rheumatic diathesis). The principal feature by which dorso-intercostal Neuralgia can be separated from myalgia is its history; viz. its non-dependence, or much less dependence, on *excessive or long-continued local muscular action* than the latter complaint exhibits. There is also a more marked *intermittence* in the neuralgic affections. Finally, though this only applies to a limited number of cases, the intercurrence of *herpes* is a decided diagnostic of the neuralgic character of the disease.

Dorso-intercostal Neuralgia is an affection of certain of the dorsal nerves. These nerves divide immediately after their emergence from the intervertebral foramina into a posterior and an anterior branch. The former sends filaments which pierce the muscles, to be distributed to the skin of the back; the latter, forming the intercostal nerve, follows the intercostal space. Immediately after their commencement the intercostal nerves communicate with the corresponding ganglia of the sympathetic. Proceeding outwards, they at first lie between the pleura and intercostal muscles; towards the angles of the ribs they pass between the two layers of intercostal muscles, and, after giving branches to the latter, give off their large superficial branches. In the case of the seventh, eighth, and ninth intercostal nerves, which are those chiefly liable to Neuralgia, the superficial branch is given off about midway between the spine and the sternum. The final point of division, at which superficial filets come off, in all the eight lower intercostal nerves, is nearer to the sternum, and is progressively nearer to the latter in each progressive space downwards. There are thus, as Valleix observes, three points of division: 1, at the inter-vertebral foramen; 2, midway in the intercostal space; 3, near to the sternum. And there are three sets of superficial branches (reckoning the posterior primary division) which make their way towards the surface near these points.

In one of its forms, intercostal Neuralgia is one of the commonest of all neuralgic affections. I refer to the pain beneath the left mamma, which women with neuralgic tendencies so often experience, chiefly in consequence of over-lactation, but also from exhaustion caused by menorrhagia, and especially from the concurrence of this cause with the preceding one. Some care must be taken to distinguish this from the mere myalgic pain, which is produced by over-working the pectoral muscles in proportion to the existing state of their nutrition, and also by the vague conditions grouped under the name "Hysteria." The latter sort of pain is more diffuse in extent, and less markedly intermittent, than Neuralgia, and its history is different: and the effect of *rest* is far more marked in the former than in the latter.

It is only of recent years that the Neuralgia which had often been observed to attend herpes zoster has been even thought of as essentially connected with the latter disease. It is to M. Notta that some of the earliest observations leading to the latter view are to be attributed. But the matter was much more fully discussed by M. Barensprung, in a paper published in 1861.[1] This author showed the absolute universality with which unilateral herpes, wherever developed, closely followed the distribution of some superficial sensory nerve, and gave reasons, which will be discussed hereafter, for supposing that the disease originates in the ganglia of the posterior roots, and that the irritation spread thence to the posterior roots in the cord, causing reflex Neuralgia. This theory will be discussed further. Meanwhile, it seems to be established, by multiplied researches, that though unilateral herpes may, and often does, occur without Neuralgia, and Neuralgia without herpes, the concurrence of the two is due to a mere extension of the original disease, which is a nervous one.

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[1] Annalen der Charité Krankenhauses zür Berlin, ix. 2, p. 40: Brit. and For. Med. Rev., January 1862.

In young persons zoster is not often attended with severe *Neuralgia*, but a curious half-paretic state of the skin, in which numbness is mixed with formication, or with a sensation as of boiling water under the skin, precedes the outbreak of the eruption by some hours, or even a day or two. Painless herpes is commonest in youth. From the age of puberty to the end of life the tendency of herpes to be complicated with Neuralgia becomes progressively stronger. The course of events is different in different cases, however. Usually, in adult and later life the symptoms *commence* with a more or less violent attack of neuralgic pain, which is succeeded, and for the time usually (though not always) displaced, by the herpetic eruption. This latter runs its course, and after its disappearance the Neuralgia very commonly returns again. In old people the *after*-Neuralgia is often distressingly severe, and most rebellious to treatment. Six weeks or two months is quite a common period for it to last, and in some aged persons it has been known to fix itself permanently, and cease only with life. In elderly subjects a further complication sometimes occurs. The herpetic vesicles leave obstinate and most painful ulcers behind them, which refuse to heal, and worry the patient frightfully, the merest breath of air upon them sufficing to cause agonizing darts of neuralgic pain. I have known one patient distinctly killed by the exhausting agony thus caused.

The foci of pain in intercostal Neuralgia are always found in one or more of the points, already mentioned, at which sensory twigs become superficial. In long-standing cases acutely tender spots are developed; not unfrequently the most decided of these are where they are too seldom sought for, namely, opposite the emergence from the inter-vertebral foramina.

(*e*) *Dorso-lumbar Neuralgia.*—The records of this affection are as yet in a state of considerable confusion. What has been done with any precision towards clearing up the history of the disease, related chiefly to the neuralgic affections of the pelvic organs in women; and to the Neuralgia of the testes in men, which will be treated of in a different place.

The principal foci of dorso-lumbar Neuralgia, when this affects external parts, are the following:—(1) the *vertebral* points, corresponding to the posterior branches of the respective nerves; (2) an iliac point, about the middle of the crista ilii; (3) an *abdominal* point, in the hypogastric region; (4) an inguinal point, in the groin near the issue of the spermatic cord, from whence the pain radiates along the latter; (5) a *scrotal* or *labial* point, situated in the scrotum, or in the labium majus.

Such is the description given by Valleix; and as I have seen but few examples of the external forms of dorso-lumbar Neuralgia, I can only rely upon his observation. The few severe cases of this kind of Neuralgia, which I have observed, have been distinguished by foci in the vertebral region, and over the crista ilii; in two of these there were also distinct foci in the spermatic cord and testicle. In one patient there was an apparent focus of pain higher up in the groin also; but this man is a confirmed hypochondriac, and his morbid sensations are so shifting as to be very unreliable in their indications.

(*f*) The next group of Neuralgias which must be described is the *crural.* This, after all, includes very few independent cases. There are very few primary Neuralgias of the crural nerve; Valleix had only seen two in his very large experience, and I cannot say that I have seen any. Neuralgia of the crural nerve is almost always a secondary affection, arising in the course of Neuralgia, which primarily showed itself in the external pudic branch from the plexus.

(*g*) The last and one of the most important and numerous groups of external Neuralgias are the *femoro-popliteal, or Sciatic.*

Sciatica is a disease from which youth is comparatively exempt. Valleix had collected 124 cases; and in not one was the patient below the age of seventeen; only 4 were below twenty. In the next decade there were 22; in the next 30; and the largest number of cases, 35, were between the ages of forty and fifty. This completely tallies with my own experience; and seems to favour the suspicion which I have formed, that the pressure exerted on the nerve in locomotion and in sitting is one principal cause of the great liability to Neuralgia which distinguishes the sciatic nerve; and this idea seems to be favoured by the further fact elicited by Valleix, that from thirty years onward the number of male is greatly higher than that of female sciatic patients.

There are three very distinct varieties of the disease, however, according to my experience. The first variety is obscure in its origin, but may be said, in general terms, to be connected with a strongly marked nervous temperament, which is indicated in the female by a tendency to hysteria, and in the male by an abnormal sensibility to nervous impressions. The subjects of this variety of sciatica are mostly below the age of forty, and have generally been liable to other forms of Neuralgia; the actual attack of sciatica is excited by some bodily fatigue or mental distress which, on other occasions, has produced sick-headache, or intercostal Neuralgia, &c. Very many of these patients are anæmic. The greater number of them are females, and in many (whether as cause or effect) there is decided amenorrhœa, and sometimes chlorosis. In this variety the pain, though chiefly affecting the sciatic nerve and its branches, is apt secondarily to invade some of the nerves which issue from the lumbar plexus. I cannot avoid the suspicion, though the proof is most difficult, that the affection not unfrequently depends on, or is much aggravated by, an excited condition of the sexual apparatus: certainly, I have observed it with marked frequency in women who remain single long after the marriageable age, and in the case of several male patients there has been either the certainty or a strong suspicion of venereal excess. The actual outbreak of pain is generally sudden, but in many instances there has been a tendency to numbness, or abnormal sensations, in the skin of the back part of the thigh, or in some part of the course of the branches of the nerve for some time previously. Like all forms of sciatica, this affection is usually obstinate, and requires assiduous and sometimes prolonged treatment for its removal; but it is incomparably more manageable than other varieties.

The second variety of sciatica occurs for the most part in middle-aged or old persons who have long been subject to excessive muscular exertion, or have been much exposed to cold, and especially *damp* cold, or who have been subjected to both of these kinds of evil influences. One must include also, I think, in this group, a certain number of patients whose age need not be so advanced, but who have been liable, along with depressing influences of a constitutional kind, to prolonged pressure on the nerve from the habitual maintenance of the sitting posture, in their business, for many hours together.

The patients who suffer from this second variety of sciatica are mostly, as already said, of middle age or more; but this statement must be understood to be made in the comparative sense which refers rather to the vital condition of the individual than to the mere lapse of years. Many of them have hair which is prematurely grey; and in some the existence of rigid arteries, together with *arcus senilis*, completes the picture of organic degeneration. In particular cases where depressing influences have been at work for a long time, or unusually active, these appearances rectify the impression we should otherwise receive from learning the nominal age of an individual; this is especially the case with persons who have for a long time drunk to excess. I am at a loss to know how Valleix and many others can have overlooked the frequent occurrence of this type of constitution among the most numerous group of sciatic patients—those between thirty and fifty years of age: unless, indeed, we suppose that many of their "robust" patients were so fresh in colour and possessed such good muscular strength, as to lead the physician to ignore the far more significant vital indications which are given by the above-mentioned appearances.

A prominent feature in this variety of sciatica is its great obstinacy and intractability. Another equally marked is the development, around one or more foci of severest pain, of spots which are permanently and intensely tender, and the slightest pressure on which is sufficient to renew the agony of acute pain: this development of tender points is far less marked in the preceding form of the disease. The places which are specially apt to present this phenomenon are as follows:—(1) A series or line of points, representing the cutaneous emergence of the posterior branches, which reaches from the lower end of the sacrum up to the crista ilii. (2) A point opposite the emergence of the great and small sciatic nerves from the pelvis. (3) A point opposite the cutaneous emergence of the ascending branches from the small sciatic which run up towards the crista ilii. (4) Several points at the posterior aspect of the thigh, corresponding with the cutaneous emergence of the filets of the crural branch. (5) A *fibular* point, at the head of the fibula, corresponding to the division of the external popliteal. (6) An *external malleolar*, behind the outer ankle. (7) An *internal malleolar*.

Another circumstance which distinguishes the form of sciatica which we are considering, is the degree in which (above all other forms of Neuralgia) it involves paralysis. By far the largest part of the whole *motor*-nervous supply for the limbs passes through the trunk of the great sciatic; it might therefore be naturally expected that a strong affection of the sensory portion of the nerve would, in a reflex manner, produce some powerful effect on the motor element. This effect is the most frequently in the direction of paralysis. Complete palsy is rare, but in a large number of cases which have lasted some time there can be no doubt that there is a positive and very considerable loss of motor power, independently of any effect which may be produced by wasting of muscles. It is of course necessary to avoid the fallacy which might be produced by neglecting to observe whether movement was merely restricted in consequence of its *painfulness*.

Anæsthesia is also a common complication of sciatica, far commoner, as I venture to think, than it has been represented either by Valleix or Notta. It is necessary, however, to be explicit on this point. In the early stages both of this form of sciatica and of the milder varieties previously described, there is almost always partial numbness of the skin previous to the first outbreak of neuralgic pain, and during the intervals between the attacks. By degrees this is exchanged, in the milder form, for a generally diffused hyperæsthesia around the foci of neuralgic pain, while other portions of the limb may still remain anæsthetic. In the severer forms it sometimes happens that, besides an intense hyperæsthesia of the skin over the painful foci, there is diffused hyperæsthesia over a greater part or the whole of the surface of the limb. But it is important to remark that both in the anæsthetic and the hyperæsthetic conditions (so-called), the *tactile sensibility is very much diminished*. I have made a great many examinations of painful limbs in sciatica, and have never failed to find (with the compass points) that the power of distinctive perception was very decidedly lowered.

Convulsive movements of muscles are met with in a moderate proportion of the cases of severe sciatica of middle and advanced life, in which affection they are entirely involuntary. They differ from certain spasmodic movements not unfrequently observed in the milder form (and especially in hysteric women), for these are more connected with defective volition, and are, in truth, not perfectly involuntary. In several cases of inveterate sciatica I have seen violent spasmodic flexures of the leg upon the thigh. Cramps of particular muscles are occasionally met with. I have seen the flexors of all the toes of the affected limb violently cramped; and in one case the patient was troubled with severe cramps of the gastrocnemius. It is chiefly at night, and especially when the patient is just falling asleep, that this kind of affection is apt to occur.

A third variety of sciatica is the rather uncommon one (so far as my experience goes) in which inflammation of the tissues around the nerve is the primary affection, and the Neuralgia is a mere secondary effect, from mechanical pressure on the nerve, which however is, apparently, not itself inflamed. I believe that these cases are sometimes caused by syphilis, and sometimes by rheumatism. It need hardly be said that this affection is essentially different, and requires a different treatment from Neuralgias in which the disturbances originate in the nervous system.

(II.) *Visceral Neuralgias.*—This most important class of diseases still remains very much unknown; but it is constantly assuming a greater consequence. The Neuralgias of viscera, of which anything can with confidence be said, are the following:—(1) Cardiac, (2) Hepatic, (3) Gastric, (4) Peri-uterine (including ovarian), (5) Testicular, (6) Renal.

It is, however, unnecessary to describe the clinical history of these disorders here, since they will be treated of under the headings of the morbid affections of the particular organs which they infest.

Complications.—This part of our subject is of the greatest interest, and the facts regarding it are, to a considerable extent, of recent discovery. If we turn to the excellent treatises of Valleix and Romberg, which appeared about a quarter of a century ago, we find a very inadequate importance assigned to the secondary affections which occur in Neuralgia. The convulsive movements of the facial muscles which occur in the severer forms of *tic douloureux* could not fail, of course, to attract attention even from the earlier times. Of the functions of special sense Valleix only mentioned *hearing* as liable to be affected. Injection of the conjunctiva he spoke of as if it were a rare phenomenon in trigeminal Neuralgia. He did not mention modifications of nutrition at all, except those of the hair; and of modifications of secretion he only enumerated lachrymation, mucous flux from the nostril, and salivation as occasional phenomena. Of disturbances of the stomach he took a more appreciative view; and he mentioned, as a remarkable fact, that he never knew facial Neuralgia caused by gastric disturbance, but had frequently observed the latter affection to occur in the course of a neuralgic attack, and apparently as the consequence of it. He gives no pathological explanation of the connexion between them.

It is to M. Notta[1] that we owe the first scientific treatment of this subject of the complications of Neuralgia. The importance of these secondary affections is particularly brought out by this author in his remarks on trigeminal Neuralgia, of which he analyses 128 cases. As regards special senses, he states that the retina was completely, or almost completely, paralysed in ten cases, and in nine others vision was interfered with; partly, probably, from impaired function of the retina; but partly, also, from dilatation of the pupil, or other functional derangement independent of the optic nerve. The sense of hearing was impaired in four cases. The sense of taste was perverted in one case, and abolished in another. As regards secretion:—Lachrymation was observed in sixty-one cases, or nearly half the total number. Nasal secretion was repressed in one case; in ten others it was increased on the affected side. Unilateral sweating is spoken of more doubtfully, but is said to be probably present in a considerable number of cases. In eight instances there was decided unilateral *redness* of the face, and five times this was attended with noticeable *tumefaction.* In one case the unilateral redness and tumefaction persisted, and were, in fact, accompanied by a general hypertrophy of the tissues. *Dilatation of the conjunctival vessels* was observed in thirty-four cases. Nutrition was affected as follows:—In four cases there was unilateral hypertrophy of the tissues; in two, the hair was hypertrophied at the ends, and in several other cases it was observed to fall off or to turn grey. The tongue was greatly tumefied in one case. Muscular contractions, on the affected side, were noted in fifty-two cases: of these, in thirteen, the contractions were in the muscles of the lip and nostril; in ten, there was tremor of the eyelid; in a great number many muscles were simultaneously affected. *Permanent tonic spasm* (not due to photophobia) was observed in the eyelid in four cases; in the muscles of mastication, four times; in the muscles of the external ear, once. *Paralysis* affected the motor oculi, causing prolapse of the upper eyelid, in six cases; in half of these, there was also outward squint. In two instances, the facial muscles were paralysed in a purely reflex manner. The pupil was dilated in three cases, and contracted in two others, without any impairment of sight; in three others it was dilated, with considerable diminution of visual power. Finally, with regard to common sensibility,—M. Notta reports three cases in which *anæsthesia* was observed. *Hyperæsthesia* of the surface only occurred in the latter stages of the disease.

Various other observers have added to this list of the secondary affections which may occur in facial Neuralgia the following:—Iritis, glaucoma, corneal clouding, and even ulceration; periostitis, unilateral furring of the tongue, herpes unilateralis, &c.

All the above complications of facial Neuralgia, excepting glaucoma, have been under my own observation, and most of them I have seen in a great many cases. Moreover, my own attention had been called independently to the subject by my own unlucky personal experience. I began, at the age of about fourteen, to suffer from attacks of unilateral facial Neuralgia in the right side (chiefly supra-orbital), which very soon assumed the type of severe migraine, such as it has already been described. A year or two later, the pains being at this time severe and frequent, there occurred a painful thickening and tumefaction of the periosteum round the brow, and also the formation of one or two dense white patches on the cornea, in the centre of which small phlyctenular ulcers appeared. About the same time, probably, there occurred a great thickening of the fibrous tissue, surrounding the upper end of the nasal duct, which caused a dense stricture of that canal. Some years later when the attacks had become much less frequent, they recurred with great severity during the prostration brought on by choleraic diarrhœa. I then first noticed that the hair of the eyebrow was whitened opposite the supra-orbital notch, and that grey hairs were thickly strewn over the right side of the head for some time after the attack; and this phenomenon has occurred after every severe attack since that time. It only lasts in intensity for a few days, and the colour soon becomes partially restored to its original tint, but *without any falling off of the hair.* The latter fact seems at first difficult of belief; but I have most closely observed the phenomenon, and have since witnessed the same thing in several patients, both of my own and other practitioners. Another nutritive modification which I have seen in my own case is the formation of a dense epithelial fur on one-half of the tongue.

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[1] Archives Générales de Médecine, 1854.

There is another complication which, so far as I am aware, was first identified by myself as having a definite relation to facial Neuralgia: viz. erysipelatoid inflammation of the tissues to which the painful nerve is distributed. Some years ago I was much surprised at observing, in a woman aged thirty-two, a patient of the Chelsea Dispensary, a most acute attack of unilateral erysipelas of the face and head, supervening on some severe and frequently recurring attacks of Neuralgia, which affected all three divisions of the trigeminus, but was most violent in the branches of the ophthalmic division. On the recurrence of the erysipelas, the acute pain subsided, but the most intense tenderness remained for some days, and pressure anywhere in the track of the nerves would re-excite a momentary spasm of pain. Since that time I have been constantly on the look-out for similar cases, and have observed a good many either in my own practice or that of others. In several instances I have seen Neuralgia of the fifth actually terminate in an affection undistinguishable from ordinary erysipelas, limited to the painful parts: in four of these cases it was limited to the side of the nose, the infra-orbital and frontal regions. But the facts bearing on a connexion between facial neuralgia and erysipelas, are by no means limited to this. In twenty-two cases which have come under my care, of patients suffering either from typical facial tic, from migraine, or from clavus hystericus, I have discovered, by inquiry, the existence of a strong tendency to erysipelatoid inflammation of the parts then affected with Neuralgia. An attack of erysipelas would be brought about in these patients, by the most trivial causes, by a slight exposure to cold winds, or, on the other hand, by unusually depressing fatigue or emotion. The majority of these patients give me a family history which showed a marked inherited disposition to neurotic affections, a circumstance which, as we shall hereafter see, is of importance.

Perhaps the most striking of all the cases which have come under my notice is one which was obligingly sent to me by Mr. Ernest Hart, and which I have already published[1] in detail. The exciting cause of the whole train of phenomena was apparently fright from an accident which there was no reason to suppose inflicted any direct physical injury. The sequence of events was: (1) abrupt cessation of menses, with hysteric depression; (2) severe neuralgia of the first and second divisions of the fifth, quickly producing iritis, with effusion of lymph; (3) erysipelas, exactly limited to the skin of the painful parts, and as it were supplanting the Neuralgia.

The concurrence of *iritis* with the erysipelas, in this case, is a most interesting fact, as showing a general tendency to paralysis of the vessels in the affected district, which will be much dwelt on in the section on pathology. The connexion of iritis with Neuralgia is a subject which, although only quite recently mooted, already assumes an extraordinary magnitude, and may yet lead to pathological and therapeutical discoveries of first-rate importance. For my own part I do not hesitate to express the belief that the very vague and ill-defined disease known, in common phrase, as "Rheumatic iritis," is destined to be almost, if not quite, banished to limbo; for, that careful observation will prove the cases so denominated to be nearly all capable of classification as "Neuralgic iritis."

The symptoms which characterise this malady are as follows. The patient first of all complains (usually after exposure to cold wind, or damp or both) of pain round the orbit, which gradually increases to a pitch of great severity, but which exhibits marked intermissions or at least remissions. The vessels of the conjunctiva, but more particularly of the sclerotic, then become injected. Last of all the iris itself becomes cloudy, and, in severe cases, actual deposits of lymph take place. I cannot hesitate to say, from careful inquiries into the past history of such patients, that this kind of affection occurs quite as frequently in persons who have never shown any distinctive rheumatic tendencies as in those who have. On the other hand, there is nearly always a recognisable history of tendencies towards neuralgic affections of one sort or another. And indeed with regard to the whole series of so-called chronic rheumatic affections of fibrous membranes, it must be remembered that there is reason to doubt whether, on careful analysis, their local symptoms can be grouped into any intelligible unity. It seems far more likely that, as the consequences of spinal irritation become more perfectly known, the whole group of such affections will be resolved into particular cases of centric nervous irritation.

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[1] Lancet, 1866, vol. ii. p. 548.

And finally it may be noted that this variety of iritis is greatly more amenable to the influence of quinine than to that of any other remedy; in fact, beyond the use of belladonna to prevent pupillary adhesion, no other treatment is required.

Herpes, as a complication of dorso-intercostal Neuralgia, has been already referred to. Although not so commonly, it may probably attend Neuralgia of any superficial nerve. For instance, the occurrence of a regular facial herpes zoster has been considered by many authors not so much a rarity as an impossibility. But various single cases have been recorded by individual observers of late years; and in a very valuable paper on unilateral herpes in the London Hospital Reports for 1866, Mr. Jonathan Hutchinson reckons up fourteen cases, including several which came under his own observation: some of them are mentioned to have been accompanied by Neuralgia of the fifth. In one of these cases, in which the Neuralgia was particularly severe, the herpetic vesicles were followed by ulcers, which left considerable scars on the forehead. I have myself seen herpes the attendant of two cases of cervico-brachial Neuralgia, in one of which the ulcerations following the vesicles were a cause of severe suffering; and in one instance of sciatica in my practice there occurred enormous vesicles, or rather bullæ, on the back of the calf, which formed most troublesome and exquisitely painful ulcers. Barensprung[1] records a similar case, in which the irritation of the sciatic was secondary to psoas abscess.

The tendency of deeper tissues to be affected in an inflammatory manner as a consequence of Neuralgia, which is specially shown in the cases of neuralgic iritis, receives every-day illustration. In fact, the painful points so universally observed in severe or inveterate cases are probably produced by a sub-acute inflammation, first of the fibrous membranes (periosteum or fascia) in contact with the nerve at points where it comes out from a deeper to a more superficial position, and further (in some cases) to all the subcutaneous tissues for an inch or two round. In one of the cases of cervico-brachial Neuralgia already referred to, a bright red painful spot, as large as half-a-crown, appeared on the outer side of the arm; there was dense thickening of tissues in this situation, and the resemblance to an inflamed syphilitic node was remarkable. The neuralgic origin was, however, unmistakeable. Among the cases of facial herpes collected by Hutchinson, there are several in which serious or even irremediable damage was inflicted on the eye by general inflammation of its tissues.

DIAGNOSIS.—The diagnosis of neuralgic affections from others which may involve pain is, on the whole, not difficult, if we are able to extract from the patient a full account of his history. The essential points for observation are:—1. The situation and direction of the pain, whether this is unilateral, whether it corresponds to the course of a recognisable nerve branch or branches. 2. Whether it is intermittent, or markedly remittent. The points of history which are most important are:—1. Whether the patient has suffered Neuralgia before, and if not, whether Neuralgias, or neurotic diseases of any kind, have prevailed in his family. 2. Whether the attack was preceded by nervous disposition, or was ushered in by distinct numbness or tingling. 3. Whether the immediate excitant appeared to be cold or damp or both, or a severe nervous shock, or a direct physical injury. 4. (If the affection has lasted some time) whether there has occurred any development of secondary tender points in the situations where, as above described, they might be expected. 5. Whether the patient has suffered from secondary affections of glands (*e.g.* lachrymation, in the case of facial pain) during the attacks, or of temporary congestion of surfaces (*e.g.* of the conjunctiva) in the same case, or from alterations of epithelium or hair, or herpetic eruptions, or erysipelatoid inflammation of the skin corresponding to the distribution of the affected nerves.

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[1] Loc. cit.

The affirmative answer to any of these questions is, *pro tanto,* in favour of the genuinely neuralgic character of the disorder; and, indeed, the union of features 1 and 2, under the heading of "observation," with one, or still more with two or three, of the "historical" facts, would be pretty well decisive in this sense.

The main source of embarrassment, in difficult cases of diagnosis, is the impossibility which we sometimes encounter of getting a clear history. This is especially apt to occur when we are called to the patient not so much on account of the primary neuralgic affection as because of severe secondary consequences that happen to have arisen. For instance, in a case of severe Neuralgia of the fifth, attended with periosteal inflammation round the orbit, or with intense conjunctivitis, and, it may be, corneitis, or even iritis, the history related is likely enough to lack explicit details of the primary affection. It is necessary to inquire very strictly whether the pain, when it first occurred, was, or was not, accompanied by tenderness on pressure; and whether this simple pain markedly preceded the organic lesions.

Another serious difficulty arises, not unfrequently, in distinguishing between true Neuralgia, and that form of pain which is vaguely called hysteric; and also between the former, and Myalgia not associated with the hysteric diathesis. The great characteristic of true Neuralgia is the limitation of the pain to the course of recognisable branches of nerves, as opposed to the diffused character both of hysteric and neuralgic pains. A history of intense hysteric predisposition may help the diagnosis in some cases, and a history of overwork done by under-nourished muscles may clear it up in others. But hysterical persons may, and sometimes do, suffer from true Neuralgia. And again, it is very common for hysteric patients to develop tender points in certain situations (especially beneath the left mamma, in the epigastrium, and at various situations along the vertebral fossæ which lodge the great muscles of the back), which bear a superficial similarity to the tender points developed in long-standing Neuralgia. The more generalised hyperæsthesia of the skin which usually accompanies these symptoms, when they are due to hysteria, will seldom be observed, however, in true Neuralgia; and the remarkable affections of volition which mostly accompany the hysteric diathesis rarely occur in Neuralgia pure and simple. A means of diagnosis between hysteric hyperæsthesia and the true Neuralgia which I have found most useful is the use of Faradisation. It has a strikingly inactive effect in the former, but acts much more slowly, or not at all, in true Neuralgia.

It is almost impossible to lay down rules of diagnosis, in this place, between Neuralgia pure and simple, and that which accidentally occurs from a nerve becoming squeezed, or otherwise damaged, in the progress of tumours or other organic diseases external to it. The reader must be referred to the diagnostic characters mentioned in the treatises on such diseases for the means of distinction.

The neuralgic pains which usher in locomotor ataxy, are highly peculiar, and their diagnosis from ordinary Neuralgia must be learned by studying the article on the former disease.

PROGNOSIS.—The prognosis of Neuralgia is nearly always an uncertain matter. The simplest case is when a clearly malarial history can be made out, and when the blood infection has not lasted too long: here we may expect a speedy cure by appropriate treatment. The least complicated varieties of traumatic Neuralgia—those in which the irritation is only kept up by some mechanical irritation (*e.g.* a foreign body lodged, or a tight cicatrix making pressure)—of course offer a good chance of cure by surgical interference. Among the Neuralgias which are more purely of internal origin, those are chiefly to be regarded as benign which occur in young subjects; and next to youth in favourable influence on the prognosis comes the fact of otherwise unbroken health. Neuralgia becomes progressively less curable in each successive decade of life, and more especially after the commencement (at whatever nominal age) of the symptoms of organic degeneration. Very formidable, in all cases, is the fact that the patient's family have been liable either to severe Neuralgias, or to other grave neuroses. And when a patient with such a family history is first attacked with a Neuralgia after he has already entered on the period of organic degeneration, his chances of complete recovery must be reckoned very small. Moreover, such a Neuralgia is not unfrequently the first warning of a degeneration of the centres, which will end with softening of the brain.

These are the fundamental points in prognosis. A less essential, but still important, class of momenta are the circumstances of the patient's life; how far, for instance, he is likely to be exposed to the hostile influences of cold, damp, and privation, with the disorders which they tend to engender; and how far there may be unavoidable exposure to the influences of mental distress, or of "the weariness of an objectless life."

Pathology and Etiology.—These two subjects, in the case of Neuralgia, are inextricably mixed; nor is it possible to discuss the one without constant reference to the other. They are so mixed, firstly, because there is no sufficient basis of anatomical fact to support a "pathology," in the ordinary sense; and secondly, because, in addition to the philosophical difficulties which always beset the construction of an etiological system, there are, in the case of Neuralgia, special obstacles to the decision as to what is "cause" and what "effect," arising from the necessity of regarding a neuralgic person as a mere offshoot of a certain family beset with peculiar tendencies, rather than as an individual who forms his own physical destiny by the manner and circumstances of his life.

Of facts tending to elucidate the morbid anatomy of Neuralgia there are very few. This necessarily follows from the rarity with which neuralgic patients die under circumstances which lead to any careful examination of the nerves and nerve-centres. Among the very few recorded cases which show anything positive is the remarkable one related by Romberg.[1] The patient was a victim to the severest form of facial Neuralgia, "of the period of bodily degeneration," such as I have described it. The Gasserian ganglion of the painful nerve was almost destroyed by the pressure of an internal carotid aneurism, the trunk and posterior root of the nerve were completely degenerated, and the atrophic process had extended, in less degree, to the nerve of the opposite side.

This case, alone, of course *proves* nothing as to the general question of the pathology of Neuralgia. But it teaches a notable fact, that the extremity of pain can be suffered in a nerve in which sensation would soon have become extinct by dissolution of the connexion between centre and periphery. It is imaginable that a not less real, but less advanced and less coarsely obvious atrophic change may have been present in every case of Neuralgia, even where dissection has failed to reveal anything amiss. It must be remembered that the microscopic study of morbid changes in nerve tissues is even now only in its infancy. It would be vain to occupy a large space in a practical treatise, with disquisitions on a subject at present so obscure as the pathology of Neuralgia; I shall therefore content myself with stating the hypothesis which appears most probable to me, and the mere outline of the reasons which incline me to adopt it.

I think it most probable that in *all* cases of Neuralgia there is either atrophy, or a tendency to it, in the posterior or sensory root of the painful nerve, or in the central grey matter with which it comes in closest connexion. The following are the heads of the argument:—

1. Neuralgia is eminently hereditary. It is constantly observed to prevail in particular families, breaking out in successive generations

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[1] Diseases of Nervous System, Syd. Soc. Trans. vol. i.

and various individuals. But what is even more important to notice is the fact that these neuralgic families are almost invariably also distinguished by a tendency to the severer neuroses—insanity, cerebral softening, paralysis, epilepsy, hypochondriasis, or an uncontrollable tendency to alcoholic excess; and very often in the various members of the same family we may observe the alternation of all these affections with Neuralgia.

2. Such hereditary tendencies in a race seem strongly to suggest a tendency to imperfection in the congenital construction of the central nervous system; so that we may imagine that certain cells and fibres of this system are, in a large proportion of that race, built, as it were, only to live with perfect life for a short term. The weak spot may be in one place in this person, in another place in that.

3. Given such a weak spot, congenitally present, all hostile influences will tell more heavily on it than on the rest of the organs. The depressing influence of cold applied to the periphery, of a wound of the trunk or branches of a nerve, of a severe shock (mental or physical) to the nervous centres generally, or of continued alcoholic excesses, will suffice to throw the imperfectly constructed cells into a state of positive disease, which may end in decided atrophy. Even in the absence of any special external cause, the depressing influence on the nervous centres produced by the great crisis of puberty, childbearing, the involution of the female organs at the grand climacteric, and still more the partial failure of nutrition which the arterial degeneration of advanced life would cause—any of these may suffice to start the local morbid process.

4. A very weighty argument in favour of the idea that central mischief is a factor in all cases of Neuralgia is the great frequency of complications, such as have been described, in which various nerve-fibres, quite distinct from those which are the seat of pain, and connected with these only through the centre, are secondarily affected.

5. Those cases in which a localised peripheral lesion is the immediate excitant also require for their explanation the assumption of a peculiarity in the individual, as one factor, and that the most important, in the production of the Neuralgia. For of hundreds of persons to whom exactly similar lesions happen every year, not more than two or three, perhaps, experience any Neuralgia; and these two or three will, I believe, be invariably found to belong to neurotic families.

6. The only cases in which the theory of congenital central imperfection appears neither applicable nor necessary are those in which a pressure, ulceration, or other lesion extending from neighbouring tissues towards the nerve, maintains a constant depressing centripetal influence which it is not difficult to suppose might impair the vitality of the posterior root, or of the central grey matter.

7. Certain influences, especially that of excessive drinking, which notoriously tends to produce degeneration of the nervous centres, are powerful predisposers to the production of Neuralgia of the inveterate

type. Moreover, the descendants of drunkards, among other evidences of an enfeebled nervous organization, are decidedly prone to Neuralgia. So frequently have I made the discovery that neuralgic patients have had drunken parents, that I cannot suppose the coincidence to be accidental.

TREATMENT.—The treatment of Neuralgia may be classified under three heads. The first division includes all remedial measures which are intended to improve the general nutrition, including that of the nervous system, or to remove any vicious condition of the blood which may impair nervous function. The second division includes the narcotic stimulant remedies. The third division comprises all the remedies which are destined to exert a direct influence upon the affected nerve.

1. Constitutional treatment.

(*a*) Under the head of *nutritive* remedies for Neuralgia, by far the most important sub-class is the series of animal fats. There is a theoretical basis for the use of these substances which it is impossible to ignore, although I have no desire, in the present state of our knowledge, to insist too absolutely upon it. In some way or other, fat must undoubtedly be applied to the nutrition of the nervous system, if this is to be maintained in its organic integrity; since fat is one of the most important, if not the most important, of its organic ingredients. But if our theoretical ideas on this point be as yet deficient in the exactness which is to be desired, there can be no doubt, I think, that the practical lessons which they would teach are abundantly verified in experience. If we take, for instance, the class of Neuralgias which are most plainly and indubitably connected with impaired nutrition—those of advanced life, and particularly the inveterate forms of facial tic douloureux—there is the strongest ground, in the results of experience, for insisting upon the value of this class of remedies. To Dr. Radcliffe belongs the merit of having been chiefly instrumental in bringing forward this therapeutical fact in this country, and it is one which I have had repeated occasions to verify. It is a very singular circumstance, which also was first pointed out by Dr. Radcliffe, that neuralgic patients are, in the majority of instances, found to have cherished a dislike to fatty food of all kinds, and to have systematically neglected its use. I have also obtained strong evidence that this is the general rule, and the reverse a rare exception. And it has several times occurred to me to see patients entirely lose neuralgic pains, which had troubled them for a considerable time, after the adoption of a simple alteration in their diet, by which the proportion of fatty ingredients in it was considerably increased.

Cod-liver oil occupies the highest rank among fatty remedies; where it does not immediately disagree with the stomach, this oil is the best fat to employ. But in other cases butter, and especially cream, may be employed with great advantage; and in fact one of the most successful examples of the treatment of Neuralgia which

I record was treated solely by the administration of Devonshire cream in increasing, and finally in very large quantities. Even the vegetable olive oil, though far inferior to animal fats as a general rule, may occasionally be used with good effect. It is necessary in many cases to make a series of trials, before we arrive at the particular form of fatty food which is best suited to the particular patient.

(*b*) The various preparations of iron are of use, so far as I know, only in cases which are marked by the existence of actual anæmia. For patients who possess well-globulated blood (as indicated not merely by the colour of the face, but by that of the mouth and tongue, especially by the freedom of the latter from teeth-markings, and by the absence of the drowsiness, *muscæ volitantes*, &c., which indicate defective blood-nutrition of the brain) I do not believe that iron treatment has any value. The carbonate, in large doses, is the best form, when iron is needed at all.

(*c*) The employment of the so-called special nerve tonics is of great use in some cases, of none at all in others. Quinine, arsenic, and zinc (in various preparations) are the only medical substances of this class which possess any solid claims to efficacy.

With regard to the efficacy of quinine there are the most conflicting opinions, except in one respect. No one doubts that in the Neuralgias which are of malarious origin this medicine, though not infallible, is extremely efficacious. It should be administered, in all cases which from their regular intermittence leave room for a suspicion that this may be their nature, in full doses (five to twenty grains) shortly before the time at which the attack of pain is expected; in fact just in the way which proves most effective in the treatment of regular ague. If after three or four doses a decided improvement is not effected, the probability is great that the Neuralgia is not malarial. Nevertheless, arsenic may subsequently be tried if other means (to be presently described) prove ineffectual.

In a certain number of non-malarial cases, also, quinine produces good effects; but there is no need, nor is it advisable, to employ it in such large doses. From two to three grains, three times a day, is the largest quantity which is likely to be of any use, if my own experience is worth anything. I know of no circumstances which indicate beforehand that quinine will be useful in non-malarial cases, *except that it seems always much more effective in Neuralgia of the ophthalmic branches of the fifth, than in other non-malarial Neuralgias.*

With regard to other non-malarial Neuralgias I share Valleix's opinion, that it is far from being frequently useful.

Arsenic is a more widely applicable remedy: for it is useful in many cases both of the malarial and of the non-malarial type. In the former it should be given, probably, in full doses, of ten minims, increasing to thirty, of Fowler's solution, three times a day. In the non-malarial forms, the ordinary tonic dose of five minims of liq. arsenicalis, three times a day, or $\frac{1}{12}$ grain of arseniate of soda in pill, with extract of hop,[1] will effect all the good which this medicine can produce. The ordinary precautions must of course be observed, as in any other case where we employ arsenic. There is one form of Neuralgia, however, which merits special mention in relation to arsenical treatment; I mean the specially neurotic form of angina pectoris. In France this remedy is extensively used for cardiac Neuralgia. I have myself seen most remarkable relief afforded by arsenic in this complaint, and an extraordinary tolerance of the system to large doses of it. Very recently, Dr. Philipp has put on record a most interesting case of the kind.[2] There are, indeed, some patients whose alimentary canal is too irritable to bear this remedy at all; but it is usually well borne, and often extremely efficacious. Arsenic may also be effectively administered by subcutaneous injection.

The preparations of *zinc*, and more especially the valerianate, enjoy a high reputation with some practitioners. It is necessary to record this fact; but I cannot say that I have ever seen any good result, which could be confidently attributed to these remedies, in Neuralgia.

(*d*) Last, among the constitutional remedies, we have to mention those which are directed against a real or presumed depravation of the blood by some special poison. Neuralgia may certainly arise from *syphilis;* but then it is probably always due to a local deposit somewhere in the course of the affected nerve. Where this can be suspected, iodide of potassium should be administered in large doses; and if this fail, the bichloride, or biniodide of mercury, in small doses. Neuralgia is said to have frequently a *gouty* origin: but the facts on which this statement rests, perhaps hardly warrant a decided opinion. They scarcely amount to more than this, that in a certain ill-defined group of cases, the subjects of which are perhaps more often than not of a gouty constitution, a form of Neuralgia occurs which yields more speedily to treatment with colchicum than to any other remedy. Twenty to thirty minims of the tincture or the wine, three times a day, will be sufficient; and if a marked good effect be not produced in two or three days, the medicine should be abandoned, or even earlier, if any tendency to weakness or irregularity of the heart's action be perceived.

"Rheumatic" Neuralgia is a phrase which, under the precautions above indicated, must still be retained, as signifying a class of cases in which inflammation of circumjacent fibrous tissues seems to cause the neuralgic pain by producing mechanical damage to the nerve. Iodide of potassium in five to ten grain doses twice or thrice daily is often useful; causing the absorption of local deposits, or rather of local proliferations of fibrous tissue. Even in cases where the Neuralgia was the primary affection, and the fibrous hypertrophy secondary to it, the local tenderness and swelling appear to be often diminished by the use of this remedy. I have never seen *colchicum* produce the slightest benefit in these cases, in which local tenderness is a prominent symptom.

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[1] Dr. Radcliffe tells me he finds that extract of hop enables arsenic to be better tolerated than when given alone.

[2] Berlin. Klin. Wochensch. 4, 1865.

2. We have now to consider the large group of narcotic-stimulant remedies for Neuralgia. In this class, I include not only the substances generally recognised as belonging to it, such as opium, belladonna, alcohol, &c., but also many others, such as ammonia, turpentine, &c., which are commonly spoken of merely as "stimulants;" and also substances which, like aconite, are ordinarily ranked either as pure "sedatives" or as "acro-narcotics." I shall not retrace here the arguments which I have given at large, in my work on "Stimulants and Narcotics,"[1] to prove that all these substances possess the common property of assisting nerve function when given in small doses, and of paralysing it when given in excess.

The narcotic-stimulant group of remedies, when administered internally or by subcutaneous injection, may be said to hold an intermediate position between the constitutional and the local agencies which we may employ against Neuralgia. On the one hand, they enter the general circulation, and pervade the organism. On the other hand, it may be suspected that in many cases their effect is produced mainly by a local action, either upon the central nuclei of affected nerves, or perhaps upon their spinal ganglia.

Indisputably, at the head of all this class of remedies stands opium. And we may consider opium, as used against Neuralgia, to be fully represented, for every useful purpose, by morphia. But the gastric administration by opiates can, after all, be only considered as *palliative*. The invention of the subcutaneous injection (which was imperfectly forestalled by the *endermic* method) has thrown quite a new light on the capabilities of opium as an anti-neuralgic. It may be confidently said that in the right use of this remedy, we possess the means of permanently and rapidly *curing* very many cases, and of alleviating, to a degree quite unknown before, the suffering caused by even the most inveterate forms of Neuralgia.

The *local* injection of alkaloids, as first systematically employed by Dr. Alexander Wood, is a proceeding which is specially applicable, in my opinion, only to a few cases. In many instances the nature of the integument at or near the point of severest pain, is such as to render the local operation inconvenient or even impossible. In the great majority of cases, especially those which are seen early, the injection may be more advantageously performed in some indifferent place, such as the loose skin over the front of the biceps muscle, or, in fact, in any place where a fold of skin can be conveniently picked up. The substance injected, if properly dissolved in a convenient quantity of fluid, quickly enters the general circulation, and, in a large majority of instances, produces just as decided an effect on the local nerve pain, as if it had been locally injected. I cannot doubt that, in the greater number of cases, the "local" injection is such only in name; the injected substance producing no effect till it has entered the absorbent vessels or the veins, and thence travelled all round the circulation to the small arteries, either of the spinal and ganglionic centres, or, perhaps, to the arteries which supply the peripheral branches of nerves. The discovery of the great utility of the plan of general, as opposed to local injection, is due to Mr. Charles Hunter, and is of the highest importance, not merely as a practical fact, but in the suggestions which it gives as to the general subject of the place of origin of Neuralgia. There is, however, a class of cases in which the local injection of morphia becomes desirable. In advanced cases, in which very great local hyperæsthesia exists, and there is reason to think that thickening and hypertrophy of the structures round the nerve has taken place, I have several times known injection at a distant point to fail, when local injection of the same substance, in the same dose, has immediately produced a marked effect; and the same thing has been recently pointed out to me by several medical men. It happens sometimes, however, that in the very cases which seem most to demand the local injection, the local tenderness makes the operation intolerably painful: in such a case I should recommend a plan which Mr. Hart introduced to my notice, viz.: that of first rendering the skin insensible with ether spray, and then injecting. As the freezing process renders the tissues quite hard, a *steel* canula to the syringe is needed to penetrate them.

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[1] London: Macmillan. 1864.

As regards the dose to be employed, I cannot but think that the received ideas are much in fault. One hears constantly of as much as half a grain or one grain, even, of morphia being employed, even at the outset. That such quantities are necessary, sometimes, where the cellular tissue injected into is already irritated and thickened, I have no doubt; and I explain it by the hypothesis that a good deal of the injected substance never enters the general circulation, nor even the vessels of the part, but lies encysted, just as is undoubtedly the case when one injects an irritant substance like pure chloroform into the cellular tissue anywhere. But I am quite certain that when injection of any non-irritant solution of morphia into a healthy cellular tissue is *neatly performed*, it is unnecessary and *even unsafe* to commence with larger quantities than $\frac{1}{6}$ gr. Both in my own practice and in that of a friend, I have known so little as $\frac{1}{4}$ gr. produce dangerous symptoms of poisoning in a person not especially sensitive to opium; and I am convinced that the activity of remedies hypodermically used is generally much underrated. I have produced all the desired effects by injection of not more than $\frac{1}{10}$ gr. in slight cases, and very rarely indeed (where the morphia is injected at an indifferent spot) do I increase the dose beyond $\frac{1}{2}$ gr. The best medium dose is $\frac{1}{6}$ gr.; and the injections should be repeated, if possible, daily, or even twice a day in severe cases. In visceral Neuralgia, it need hardly be said, we are obliged to be contented with injection at an indifferent spot; yet (as *e.g.* in ovarian Neuralgia) we sometimes produce excellent effects.

Next to opium in value, amongst the stimulant narcotics, is *belladonna* and its alkaloid *atropia*. The value of belladonna, as given by the stomach, is confined pretty much, according to my experience, to painful affections of the pelvic organs, on the sensory (as notoriously in the motor) nerves, of which it seems to have a special influence. In doses of $\frac{1}{6}$ gr. to $\frac{1}{2}$ gr. of the extract, it will frequently relieve ovarian dysmenorrhœa, as also some forms of superficial lumbo-abdominal Neuralgia. But by far the most important use of belladonna is by the subcutaneous injection of atropia. From the $\frac{1}{120}$ up to the $\frac{1}{30}$ of a grain is about the range of doses for adults; and I can confirm the statements of Mr. Hunter that by repeated applications of this treatment, even very severe and inveterate Neuralgias are often greatly relieved and sometimes cured. It is a question whether there is not less tendency to relapse after this treatment than after that by morphia. On the other hand, I have met with more than one person in whom it has been found impossible to give a dose sufficient to relieve the pain without producing distressing head symptoms.

Next in value to morphia and atrophia comes Indian hemp, which has been especially brought forward by Dr. Reynolds. A *good* extract of this, in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ grain or (rarely) 1 grain, given in pill, is very effective in some forms of Neuralgia, particularly in clavus hystericus and migraine. Even in the severest and most intractable forms it often palliates greatly. It should be given every night, whether there be then pain or not.

Muriate of ammonia is an excellent stimulant remedy in migraine and clavus, and in some cases of intercostal Neuralgia. It should be given in 10 to 20 gr. doses. In cases of suspected hepatic Neuralgia I have also found it very useful; and I believe that its action on the liver (in disorders of secretion) is through the nervous system entirely.

Sulphuric ether, which in the severer forms of superficial Neuralgias is of little or no effect, is supremely useful in certain visceral Neuralgias. It sometimes relieves gastralgia, and Neuralgia of uterine or ovarian origin, with magical rapidity. But it is still more valuable in the most purely nervous form of angina pectoris. I have now under my care a case of this latter affection, which I am convinced would have ended fatally long since, in one of the agonizing attacks of spasmodic heart-pain, but for the discovery that, by taking a spoonful of ether immediately on its commencement, the patient can greatly mitigate the attack. This patient had tried arsenic, but from the irritability of his intestinal canal, could not take it. The same dose of ether has continued to produce the same happy effect on each occasion of its use for the last three years.

Aconite, in the form of Flemming's tincture, is of very great use in some forms of Neuralgia, especially in that kind of ocular Neuralgia, with secondary inflammation, which is so frequently called rheumatic iritis. But, unfortunately, it is a very uncertain remedy in one respect: with some persons it produces nausea, burning in the throat, and a sense of cardiac depression, with doses which are quite harmless to other patients. In a case where I recently employed it, in only three-minim doses every six hours, I was compelled to abandon it after the third dose, from the intensely depressing effect which it produced.

The oil of turpentine is a remedy which enjoys, or enjoyed, considerable reputation for its effect in a certain class of cases. In the more obstinate forms of sciatica it is at least worth a trial, although it is commonly very disagreeable to the patient; ten minims, three times daily, is the proper dose.

Still, after the enumeration of all the narcotic-stimulant substances which have been, and many more that might be, named, it would be idle to pretend that any of them are to be compared, for wide and general efficacy, to the subcutaneous use of morphia and atropine, and the internal use of Indian hemp in small doses.

I have reserved to the last, under the head of Stimulant Narcotics, what must be said about alcoholic drinks. There can be no question about the power of alcohol to relieve neuralgic pains; it is as distinct as that of opium. But the dangers of prescribing it as a remedy are very great, since the patients cannot always be induced to use it in the strictly medical manner in which alone it is safe. Too often, instead of employing it in the moderate stimulant doses which really are of service, they accustom themselves to drowning the pain with a large narcotic dose, and thus they contract a liking for the oblivion of drunkenness. It is of much consequence, where this is possible, that they should be forbidden to take alcohol otherwise than at meal-times. If once they are induced to take it for the mere relief of acute pain, there is great danger that they will drink to excess. I am, nevertheless, convinced that a fixed daily allowance of wine or brandy (beer more rarely agrees), which shall contain not more than one ounce of absolute alcohol, is a decided help to recovery from every form of Neuralgia; and in the case of persons of firm character, who can be trusted to exercise self-control, a larger quantity than this may sometimes be allowed. Without pretending to speculate on the physiological reason for it, I must add my testimony to the fact, which has been observed by Dr. Radcliffe, that *saccharine* liquors and saccharine foods, except in very moderate quantities, decidedly disagree with neuralgic patients.

3. We come now to consider the external remedies for Neuralgia. Incomparably the most valuable of these is the use of so-called counter-irritation; that is, the application of various irritants to the skin. Valleix comes to the conclusion that there is no one remedy which approaches *blistering* in value, and (putting aside the recently discovered hypodermic treatment) that saying remains absolutely true at the present day. It is to be observed that Valleix latterly always employed the milder form of the flying blister. Such an application as this to the foci of pain must, if we consider it, be supposed to excite a directly stimulant effect upon the painful nerve. This kind of blistering, and the analogous use of mustard plasters, have always yielded good results, in my experience, solacing even when they did not cure. And in numerous early cases one or two flying

blisters, applied successively over different points in the course of the painful nerve, have at once and permanently arrested the disease. It is a remedy which ought always to be tried in cases of any severity, especially if the subcutaneous injection of morphia and of atropine has failed. There is one method of blistering which I have recently tried with great success, viz. the application of a blister close to the spine, as nearly as possible opposite the intervertebral foramen from which the affected nerve issues. The effect produced is, I suppose, a reflex stimulation through the posterior branches. This method is of course not so applicable to Neuralgias of the fifth as to those of spinal nerves. Yet even in these, blistering of the nape has sometimes appeared to do marked good—through the occipital nerve, I presume.

The application of various stimulating liniments and ointments to the skin of the painful parts is sometimes very useful. Of these the use of chloroform diluted with seven parts of oil or soap-liniment is by far the most efficacious. This produces no anæsthesia, but a mild stimulation. Strong *counter-irritation* may be produced by the use of tartar-emetic or of veratrine ointment.

Electricity.—The efficacy of various forms of electricity in Neuralgia is a large subject, and as yet, it must be owned, only very partially cleared up. The comparative merits of Faradisation and of the continuous current are hardly settled. But the weight of testimony is now in favour of the belief that in the majority of instances the continuous current is the most valuable.

As regards one or two points, one may speak with some confidence. In the first place I may say, after extensive trials of the ordinary rotatory (magneto-electric) machine for the *induced* current, that this method of treatment is most unsatisfactory. I have never seen it produce, indisputably, good effects. Secondly, as regards that form of *continuous* current which is generated by Pulvermacher's chains, I am reluctantly obliged to give up the hope of doing any real service with it in Neuralgia, however great its utility is in other diseases. As is remarked by Dr. Althaus, the current generated by these chains is too irregular, and their activity is too soon exhausted for us to get a sufficiently uniform dose of electricity applied continuously for a definite period by their means.

It appears probable that we shall ultimately find that for neuralgic affections of all kinds the most useful form of electrical treatment is by the continuous current generated from a Bunsen's or a Daniell's battery; and that the three principles on which we must act in its use are:—1. The maintenance of the current, with only a very few breaks, for a considerable time. 2. The application of the positive pole over the seat of pain. 3. The employment of a very low-tension current. I am informed by Mr. J. N. Radcliffe, whose experience in this matter is very large, that the use of this mode of electrization in Neuralgia is as yet, in his opinion, only beginning to be developed, but that it promises to effect great things. In short my present opinion as to the value of electricity in Neuralgia may be thus expressed: that as used, up to the present time, it has achieved no results which entitle it to more than a third or a fourth rate place among remedies; but that if the desideratum of a low-tension continuous current, which can readily be applied for long periods together, can be obtained by means of apparatus of moderate portability and cheapness, it is probable that we may obtain that which will equal or exceed in value any of the remedial measures which are at our disposal.

A few words must be given to the rather uninviting subject of the surgical treatment of inveterate Neuralgia. The section of a neuralgic nerve, or rather the excision of a piece, is still, I suppose, to be reckoned among the measures which it may be occasionally justifiable to employ. Nothing, however, either in the two cases of its use which I have seen, or in the records of similar operations, would lead me to recommend it in any case. The relief given is nearly always very transient; and, indeed, the nearly infallible certainty with which the pain returns in the central end of the divided nerve is only what I should expect from the many considerations which point to the central origin of the nerve as the most peccant part. With such remedies in our hands as the subcutaneous injection of morphia, &c., I cannot see that we need to be tempted to perform such an operation for the sake of a temporary alleviation.

The removal of any distinct source of peripheral irritation by surgical means is quite another matter, and may be highly proper and necessary. Yet even here it is always necessary to calculate whether the shock of the procedure itself may not be injurious; and it will be desirable before inflicting it to fortify the system, as far as possible, with tonics; and sometimes to diminish the shock, not merely by giving chloroform, but by prolonging the chloroform narcosis by subcutaneous injection of a large dose of morphia. This precaution is especially advisable where we extract one or more carious teeth, which may seem to be keeping up neuralgic pain. Too often we find that the extraction has been in vain; and then, unless some such precautions have been taken, it may be discovered that the shock has aggravated the Neuralgia.

A most important subject, with which I may conclude these remarks on treatment, is the employment of suitable *prophylactic* measures. First, as regards nutrition; it is absolutely necessary that this should be as abundant as may be possible without deranging the digestion. It must also contain a liberal allowance of fatty matters; no amount of dislike on the patient's part—and they often show great dislike—should induce the physician to give up this point. If one form of fat cannot be tolerated, another must be tried; perseverance will, I believe, always bring success; and the effect of an improvement of this kind in the diet will rarely fail to tell upon the constitution, rendering the nervous system less sensitive to the ordinary exciting causes of neuralgic pain. Equally important is the avoidance of exposure to cold and damp air with insufficient clothing, for cold is much the most frequent immediately determining cause of neuralgic attacks. Flannel under-clothing, thick veils for the face, &c., are quite as important as any direct remedies. It cannot be doubted that everything which tends to set up the habit of pain, directly tends also to aggravate that obscure vice of the organism on which the disposition to Neuralgia depends, and *vice versâ*. Physical exercise must be so regulated that it may improve nutrition without inflicting severe fatigue. And as regards mental influences, which, unfortunately, are often beyond control, one can only say, that the two extremes, of a specially laborious and exciting life, and an existence spent in the dreary monotony of idleness, are equally hurtful.

In the foregoing article I have followed the plan also adopted in my article on Alcoholism; namely, of stating my own view of the subject connectedly, and without pausing to answer all the statements and opinions of the numerous writers who differ from me. The necessary limits of a work like this "System of Medicine," makes it almost impracticable for an author to follow any other course with success, if he happens to hold a view of his subject which conflicts with, or differs from, the views of well-known authors on a considerable number of points. But the following selected list of the more important treatises will enable the reader to study the questions connected with this disease from every point of view. It has been my purpose to bring out clearly and consistently that view of Neuralgia which seems warranted by the majority of the facts recorded by others or observed by myself; and the result has been that I have given much prominence to the arguments for the existence of an element of organic change in the *centres* in all true Neuralgias. Those who desire, however, to hear all the arguments which can be urged for a chiefly or solely peripheral origin of Neuralgia will find abundant material in the undermentioned treatises: Trousseau, "Névralgie Epileptiforme," vol. i. of his "Clinique Médicale," 2me Edit.; "Névralgies," vol. ii. of the same work (Trousseau's insistance on the constant presence of a painful "point apophysaire," seems to me an overstatement; but it is still more strange that this author should think its constant presence could consist with a peripheral origin of Neuralgia); Beau, Traité des Névralgies, Arch. de Méd. 1847; Brown-Séquard, Lectures on the Therapeutics of Nervous Diseases, Lancet, 1866, vol. i. (see also his Lectures on the Physiology and Pathology of the Central Nervous System, 8vo. Philadelphia, 1860). Of authors who allow at least a large share in the production of many cases of Neuralgia to the centres, are Teale, Treatise on Neuralgic Diseases, &c. London, 1829; C. Handfield Jones, on Functional Nervous Disorder, London, 1864; also Lumleian Lectures, Med. Times and Gaz. 1865, vol. ii. But the most suggestive and important treatise, and one which has been unaccountably neglected, is the Observations on the Functional Affections of the Spinal Cord, by William and Daniel Griffin, London, 1834. I have, in the text, given Valleix just credit for laying the foundation of the current knowledge respecting Neuralgia; but it must be allowed that in the work of the Griffins, which is little known, there are the germs of a great improvement of that knowledge. Of essays which illustrate the serious secondary complications which may attend Neuralgia, the following may be mentioned, besides the treatises of Barensprung, of Notta, the work of the Griffins, and other papers already specified:—Schiff, Hyperæmia of the Eye, Ulceration of Cornea, &c. after a Wound of the Superior Maxillary Nerve; Untersuch. p. 116: Allcock, Disease of the Eye from Injury to the Infra-orbital Nerve; Todd's Cyc. of Anat. and Physiology, vol. ii. p. 132. A great many cases also are quoted in Handfield Jones's Lectures on Functional Nervous Disorders, already cited.

It is only just to Dr. Handfield Jones to acknowledge that he has long advocated the opinion that nerve-pain is invariably, and in all its phases and consequences, an expression of debility of function; an opinion which has been strongly expressed also by myself not only in the present article, but in many other papers.

LOCAL PARALYSIS FROM NERVE DISEASE.

By J. Warburton Begbie, M.D., F.R.C.P.E.

There can be no doubt that for a lengthened period, and till a comparatively recent date, the attention of pathologists was too exclusively directed to the great nervous centres in explanation of the causes of nearly all nervous disorders, including paralysis. So much so was this the case as fully to justify the language employed by the late Dr. Graves, of Dublin. "If," says he, "you examine the works of Rostan, Lallemand, Abercrombie, and those who have written on diseases of the nervous system, you will find that their inquiries consist in searching after the causes of functional changes, either in the cerebrum, cerebellum, or spinal marrow, forgetting that these causes may be also resident in the nervous cords themselves or their extremities, which I shall call their circumferential tracts."[1] Since 1843, however, when the first edition of Graves's lectures appeared, it has been satisfactorily determined by physiological investigation and by the careful observation of disease in numerous examples, that paralysis, or the loss of the power of motion, may result from one or other of two causes. It may depend either on a central nervous lesion, that is, a lesion of the Brain or Spinal Cord, or on an abnormal condition of a particular nerve in some part of its course. It is with the latter, as giving rise to a local form of paralysis, that we are now exclusively concerned. We are abundantly familiar with the effects of mechanical injury as applied to nerves. When a nerve is cut across, there results immediately a paralysis of the parts below the section supplied by that nerve. Further, if a nerve be included in a ligature, or subjected from any cause to much pressure, a similar result is produced. The paralysis of the arm caused by pressure on the axillary plexus of nerves, is an excellent and familiar illustration of injury so occasioned. It is thus described by Dr. Todd:—"A man gets intoxicated, and falls asleep with his arm over the back of a chair; his sleep under the influence of his potations is so heavy, that he is not roused by any feelings of pain or uneasiness, and when at length he awakes, perhaps at the expiration of some hours, he finds the arm benumbed and paralysed. It generally happens that the sensibility is restored after a short time, but the palsy of motion continues. Cases of this kind sometimes derive benefit from galvanism, but if the pressure which caused the paralysis has been very long continued, they seldom come to a favourable termination. Nerve-tissue is one which never regenerates quickly, and seldom completely, so that great or long-continued lesion of its structure is not likely to be removed."[1] Although by no means so distinctly witnessed as the result is, in the class of cases now referred to, there seems no reason to doubt that, equally with mechanical injury, interference with the proper nutrition of nerves may lead to forms of local palsy. Illustrations of such occurrences will be adduced, more especially when directing attention to one of the most interesting of all the varieties of local paralysis, namely facial palsy. Again, familiar as we are with the action of various poisons—such as alcohol, opium, chloroform—on the great nervous centres, and on the same portions of the nervous system of certain poisons formed in the living body, as urea, and the morbid materials in rheumatism and gout; having also important knowledge regarding the influence which is exerted on the nervous and muscular systems generally, but especially on the nerves and muscles of the upper extremities by the poison of lead, we cannot hesitate to account, in a manner closely similar, for the other forms of local paralysis which from time to time present themselves to our notice.

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[1] Clinical Lectures on the Practice of Medicine, Lecture xxxiii.

Dr. Todd alludes to cases of local paralysis occurring in states of the constitution which, if not rheumatic, are at least allied to it, and associated with imperfect action of the kidneys. "Of this," he says, "the following affords a good example:—A medical man, ætat. 53, extensively engaged in practice in the county of Bucks, applied to me in August 1847, with complete paralysis of the deltoid muscle. He was a stout, full man, tall, of large build, and very active in his habits; fed well, and drank beer, but not to excess. He had been subject to a shifting neuralgia of the scalp, and to a discharge from the right ear, where he thought the tympanic membrane was destroyed; he was deaf on that side. Six weeks before he came to me he suffered from pain in the left side of the neck and shoulders, followed by complete paralysis of the left deltoid muscle and weakness of the whole arm. On examining, I found a total inability to raise the left arm to a right angle with the trunk, or to perform any of those actions which are usually effected by the deltoid muscle, which was very much wasted. He could, however, grasp perfectly with the left hand, and execute all the other movements of the arm and fore-arm. There was some degree of numbness of the arm. There were no symptoms distinctly referable to the head. His tongue was coated; appetite good; the discharge from the ear had ceased. The urine was pale, of low specific gravity, and contained albumen in small quantity. I viewed the case as one of local palsy, connected with a deranged state of system, rheumatic or gouty. I regulated his diet, and gave him small doses of the mineral acids. After a fortnight of this treatment he improved considerably, and could raise his arm slightly. The albumen in the urine had much diminished: and crystals of lithic acid were precipitated. He was now ordered three grains of iodide of potassium, with ten minims of liquor potassæ thrice daily. He only followed this treatment for ten days, as the iodide of potassium purged him. Still, he was improving. I continued the liquor potassæ, and advised galvanism to the muscle. This plan was diligently pursued for a fortnight, at the end of which time he had so far improved that he could raise his arm nearly to a right angle,—he could put on his coat, and tie his cravat; and in three weeks more he was quite well. All signs of albumen had disappeared from his urine."[1] The writer's experience has furnished cases bearing a remarkable resemblance to the one now quoted. He calls to remembrance more especially that of a young and plethoric as well as highly rheumatic female, who suffered from paralysis, succeeding severe pains of the left lower extremity, and in whom a plan of treatment which secured the copious discharge of urine, previously much diminished as well as disordered, and free action of the skin, proved eminently successful in removing the palsy of the limb. Besides the gouty and rheumatic poisons, it is well to keep in view the very decided action of the syphilitic in inducing this among other local disorders. No one calls in question the injurious effects which are capable of being produced on the nervous centres by the syphilitic poison; there is, however, good reason to believe that some local palsies are thus created. The writer has been able to trace the occurrence of paralysis of the portio dura, of paralysis of the third pair, as shown by a marked ptosis; and also of palsy of the limbs, slight although threatening, to the same cause, when neither brain nor spinal cord appeared to be implicated. And it is probable that the experience of many physicians has not been dissimilar to his own, in finding the iodide of potassium administered in large doses, and steadily persevered with, a most useful remedy in such cases, relieving the palsy as effectually as it is so frequently the means of doing, the neuralgic and wearing-out head-ache, or the painful node on the shin bone, which are evidently due to the same cause. Allusion has been made to the influence of direct pressure external to the body, in producing such injury of nervous structure as leads to a form of local paralysis. Palsy thus induced is generally merely temporary in duration. Tumours within the body, involving nerves, are frequently the direct occasion of local palsies. No more interesting variety of such palsy exists than that which is due to the interference with the recurrent or motor laryngeal nerve produced by an aneurism of the arch of the aorta, or by a cancerous mediastinal tumour. Well-marked atrophy of the muscles of one side of the larynx has under such circumstances been found. The dyspnœa, which is induced by the implication of the vagus, or as sometimes happens of the phrenic nerves in strumous or tubercular tumours, is abundantly recognised since the writings of Risberg and Ley. There seems reason to believe likewise that pressure upon or other injury of some parts of the sympathetic nervous system may occasion local palsies. Of this the paralysis of the radiating fibres of the iris caused by cutting the sympathetic in the neck in Budge and Waller's experiments, but especially a similar contraction of the pupil to that physiologically produced, due to the pressure of an aneurism projecting into the neck or malignant tumour similarly situated, are now quite familiar to the physician.

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[1] Clinical Lectures on Paralysis, certain Diseases of the Brain, and other Affections of the Nervous System, Lecture i.

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[1] Loc. cit. p. 72.

Attention will now be directed to some of the more important varieties of local palsy dependent on nerve disease, and first to *Facial Palsy.* This most interesting local paralysis is known under different names, of which the more commonly employed are *Facial Hemiplegia, Histrionic Paralysis, Bell's Palsy,* and *Paralysis of the Portio dura.* Occurring as it usually does on one side of the face only, nothing can be more striking than the peculiar features of the disease. This is owing to the palsied condition of a few or all of the superficial muscles—the muscles of expression—on the affected side, and the heightened antagonism of muscular action on the unaffected side. The patient cannot knit the forehead,[1] neither can the eyebrows be raised or drawn together. The eye remains open, as the power of closing the lids is lost, and their blinking movement no longer exists. This open condition of the eye, seen both in walking and sleeping, and which is due as well to the increased action of the levator palpebræ muscle as to the palsy of the orbicularis palpebrarum, is a characteristic, it has indeed been styled a pathognomonic, feature of facial palsy.[2] The ala nasi is dependent, and on full inspiration on smelling or blowing the nostrils there is no expansive movement. The angle of the mouth hangs down. Further, the patient cannot whistle, for he is unable to purse up his mouth for that purpose, and for the same reason he can neither spit, nor can he distend the buccal cavity with air, or blow wind from the mouth. Pronunciation of labials is notably impaired. The saliva and fluids frequently trickle from the mouth. In mastication portions of food are apt to collect between the cheek and gums, as the support of the lips and cheeks necessary for its proper performance is lost. Let the patient laugh, cry, sneeze, yawn, or be the subject of any violent emotion, and the distortion of the features becomes much more conspicuous, the face being forcibly drawn to the sound side. Motionless and void of expression is the one side, contrasting in a very remarkable manner with that on which intelligence remains visible and power of movement unaltered. Trickling of the tears down the cheek, owing to the immobility of the lower eyelid, with consequent dryness of the corresponding nostril, and redness of the conjunctiva, it may even be severe conjunctivitis, determined by the operation of cold, dust, or other external influences on the constantly exposed eye, are among the accompanying phenomena of this palsy.

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[1] In alluding to the smoothness of the brow in the aged, who are affected by facial palsy, owing to the disappearance of all wrinkles, Romberg facetiously observes, "für alte Frauen kein wirksameres Cosmeticum existirt."

[2] "The leading character of cases of facial palsy," writes Dr. Todd, "is the inability to close the eyelids, from paralysis of the orbicularis palpebrarum : this is the pathognomonic sign which determines the peculiar nature of the palsy, and distinguishes it from the most serious form of facial palsy, which is dependent on disease of the brain and palsy of the fifth or third nerve." (Clinical Lectures, Lecture iv.)

To Sir Charles Bell we are indebted for pointing out the true nature of this affection. He showed that one nerve only was involved, that the muscles governed by the portio dura of the seventh pair were alone affected, that strictly it is a local palsy. The sensibility of the face is usually unimpaired; a slight affection of the filaments of the fifth may, however, cause a little facial pain, but that is to be accounted rare. In instances of long-standing facial palsy, Romberg has drawn attention to the relaxed and flaccid condition of the skin covering the affected muscles, while Dr. Todd has insisted on increasing flaccidity of the cheek, and especially a rapid development of that condition, as a symptom of unfavourable omen as regards the patient's prospects of recovery. But while this form of local palsy is clearly dependent on lesion of one nerve only, there is reason to believe, as Romberg has more particularly shown, that its features are subject to modification, according to the precise seat of the disease. That may be *peripheral* or *central.* Not only so, but the diagnostic marks may vary under the former head, according as the superficial distribution of the portio dura, or the nerve as it passes through the temporal bone, or the nerve within the cranium and near its central origin, is affected. Viewing these very briefly in their order, it may be remarked—that, *facial palsy, due to an affection of the superficial distribution of the nerve,* is generally met with as the result of exposure to cold.[1] "A very common cause of this palsy," writes Dr. Todd, "is the influence of cold; as by exposure at an open window, in a coach or railway carriage, to a current of cold air."[2] "A blast of cold air on one side of the face," remarks Dr. Graves, "has been known to cause paralysis and distortion of several months' duration."[3] External injuries, such as blows on the cheek and surgical operations on the face, have been followed by this form of local palsy. Of the cases which occur, there are not a few in which no traumatic cause can be found, neither can any marked exposure to cold be traced. In such circumstances it is proper to make a very careful inquiry into the condition of the general health of the sufferer, when it is not unlikely that the connexion of the palsy with a gouty or rheumatic taint may be satisfactorily established. Dr. Todd, alluding to the dependence of periodical neuralgic affections on the determination of some poison to a particular nerve, as the paludal poison or some matter generated in the system, expresses the opinion that morbid matters may affect a motor nerve just as they affect a sensitive, causing in the former case paralysis, as in the latter they determine neuralgia.

Facial Palsy caused by an affection of the portio dura in its passage through the temporal bone.—The connexion of this paralysis with local strumous affections in children is well known. These may be simple and easily remediable, as for example the parotid and more general glandular enlargements consequent on measles, scarlatina, and other disorders; but of much more serious nature is the otitis resulting in caries of the petrous portion of the temporal bone. Here the palsy is associated with deafness, and very probably also with purulent discharge from the meatus. Direct violence, likewise, as in a case related by Sir Charles Bell, in which a pistol-shot through the ear had splintered the bone, and torn the nerve in its osseous canal, may of course determine the palsy. The diagnosis of the disease or injury affecting the nerve, in its passage through the bone, rests, according to Romberg, not only on the co-existence of such phenomena as otorrhœa, removal of necrosed portions of bone, perhaps of one or other of the small bones of the ear, and deafness,—symptoms which are not likely to occur in cases of simple peripheral facial palsy,—but, further, upon certain peculiarities in the observed paralytic phenomena. One of these is the diminution of taste on the side of the tongue corresponding to the palsy, another is a unilateral paralysis of the velum palati. On the latter point the statements of writers have been very contradictory. Romberg remarks that in four patients afflicted with facial palsy he has noticed the paralysed condition of the velum palati, the uvula, having a slanting direction, being arched and the tip pointed to the paralysed side. While failing to offer any explanation of the peculiar position of the uvula, Romberg evidently attaches very great importance, in a diagnostic point of view, to the palsied condition of the velum, and the marked curving of the uvula; concluding from their existence, that the seat of the disease *must* be in the petrous portion of the temporal bone. And he again emphatically repeats when the disease is in the peripheral distribution of the nerve, the velum is not affected, "wovon ich mich in vielen Fällen überzeugt habe." It is the implication in the diseased condition, of whatever nature that may be, of the nervus petrosus superficialis major, of Arnold—which takes its origin from the knee-shaped bulb on the trunk of the portio dura as it lies in the Fallopian aqueduct, and which communicates with Meckel's ganglion, whence the muscles of the palate derive their nerves,—that in the view of Romberg causes the displacement of the velum and uvula. Dr. Todd, while admitting the occasional occurrence of this phenomenon, combats the notion of Romberg, and maintains that undoubted instances of disease of the aqueduct, causing paralysis of the nerve, are met with, in which affection of the velum does not exist. In his own experience the symptom in question was of very rare occurrence, and he regarded it as a coincidence. Since the publication of the views of the authors now referred to, the paralysis of the palate in facial palsy has received renewed attention from M. Davaine and Dr. Sanders. The former recorded one case of unilateral paralysis of the palate, in connexion with facial palsy of right side, observed by himself, and has commented on several instances furnished by Romberg and others. His description of the phenomena he observed is given as follows :—"The velum palati is not regular; the arch formed by the right anterior pillar is less elevated than the left. The posterior pillar of the same side descends directly downwards, without being curved like that of the other side. The uvula is bent like a bow; its point is directed forwards and towards the paralysed side, while its base is carried a little towards the sound side. The patient's voice is slightly nasal."[1] Dr. Sanders, in a valuable paper,[2] gives an interesting case of paralysis of the velum in connexion with facial palsy of the right side, and enters at some length into a consideration of the mechanism of the deviation of the palate. Dr. Sanders is satisfied that a partial hemiplegia of the palate does exist in connexion with facial palsy, and, like it, is dependent on affection of the portio dura. He believes that this form of palatal palsy consists in a vertical relaxation or lowering of the corresponding half of the velum palati, with diminished height and curvature of the posterior palatine arch, on the paralysed side, and that it is due to paralysis of the levator palati,—that muscle and the azygos uvulæ, also supplied by the seventh pair, being the only muscles affected. Among several conclusions at which Dr. Sanders has arrived, the following appear to be specially important: that the partial paralysis of the velum in facial palsy, due to implication of the levator palati muscle, is by no means so rare as palsy of the velum (hitherto not accurately described) has been generally supposed, and that the prognosis is not necessarily rendered more unfavourable in facial palsy when the palate is implicated.

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[1] Some writers speak of facial palsy as specially a disease of northern climates. Thus Joseph Frank, after alluding to the collection of cases by various authors, remarks, "Nosque plurima exempla vidimus. Morbus iste in regionibus septentrionalibus tam communis est, ut spatio quindecim annorum viginti duo mihi obvenerint exempla." (De Paralysi, Praxeos Medicæ Universæ Præcepta.)

[2] Loc. cit. p. 69.

[3] Loc. cit. p. 380.

The lesion in facial palsy may exist at the cerebral origin of the seventh pair of nerves. We are not, however, called upon to consider this variety of facial palsy: suffice it to say, that its existence may be determined, and the differential diagnosis between it and the other forms—already briefly considered—established, by the occurrence, sooner or later, of symptoms due to the implication of other nerves, such as deafness, strabismus, ptosis and anæsthesia. While either the presence of inflammatory products, or apoplectic extravasations in the vicinity of the pons Varolii, may be the precise lesion which gives rise to the palsy, the probability is that, in such cases, a tumour of one nature or other, and subject to gradual extension, exists.

The duration of facial palsy is subject to considerable variety, according to the precise seat and nature of its determining lesion. Dr. Todd remarks that "it rarely, if ever, lasts a shorter time than ten days, whilst it very often extends to as many weeks; perhaps three or four weeks may be assigned as an average duration for the non-traumatic cases;" and Romberg warns us not to expect its duration to be brief. It is in those cases which have been evidently connected with rheumatism that he has found the paralysis least enduring.[3]

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[1] Gazette Médicale de Paris. 1852.

[2] Edinburgh Medical Journal, August 1865.

[3] "Die Dauer der mimischen Gesichtslähmung ist selten kurz. Am kürzesten fand ich sie bei der rheumatischen : doch habe ich sie auch hier in günstigen Fällen nur selten

The writer has seen simple cases of the disease, in so far as their cause was concerned, lasting a very lengthened period, many months, and even a year.

It is incumbent on the physician to be very careful in offering an opinion as to the prognosis in cases of facial palsy: that must always be founded on a consideration of the probable cause. Those cases are nearly certain to terminate favourably in which cold or rheumatism is to be looked upon as the determining agent. On the other hand, when the palsy has been due to mechanical injury the prognosis cannot be favourable, and this very specially in those instances where a division of the nerve has been caused. We cannot be too careful in the expression of our opinion in cases characterised by nerve disease within the temporal bone. The records of medicine contain reports of such, which have given rise to meningeal inflammation, intracranial, even cerebral and cerebellar, abscess and death.

If prognosis is to be guided by a just consideration of the causes, so also is the treatment of facial palsy when amenable to cure. The remedial measures at our disposal may be conveniently classed under the heads of internal and external agents. In the use of the former, regard should always be had to the diathetic condition of the patients,—rheumatic, gouty, strumous, syphilitic, anæmic, or suffering from the injurious influence of a paludal poison. We are disposed to think that this is one of the forms of local palsies in which the loss of power may be due to changes in nerve structure determined by neuritis. In such examples, and still more so if there be reason to conclude that a syphilitic taint is in existence, iodide of potassium will prove a most serviceable remedy. We have ourselves found it to be so. The iodide should be administered in doses of five grains twice or thrice daily, simply dissolved in distilled water. The efficacy of the remedy is secured by its being administered while the stomach is empty, but food may be taken very shortly thereafter. Should a rheumatic or gouty habit be found in connexion with the palsy, alkaline remedies, colchicum, and lemon-juice may exert a beneficial influence, and so probably will quinine or arsenic in the not unknown examples of the disease allied to intermittent fevers. Mercury in the form of blue pill has been extolled by several practitioners. Sir Thomas Watson counsels the exhibition of mercury "so as just to touch the gums," adding, "I should always take this precaution, lest any effusion of lymph should cause abiding pressure on the nerve."[1] Iron is likely to be useful when an anæmic condition of the system exists. The muriate of lime, the iodide of iron, and cod-liver oil, are available remedies when a strumous cachexia obtains. The writer can bear a decided testimony to the therapeutic value of strychnine as an internal remedy in one long-existing instance of the disease, which had bid defiance to the more

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unter sechs Wochen wahrgenommen, einmal sah ich die Heilung innerhalb acht, ein andermal in vierzehn Tagen."—P. 664.

[1] Lectures, vol. i. p. 563.

ordinary remedies; he cannot, therefore, coincide in the observation of Dr. Todd, that "strychnine is of no use in such cases."

As to external remedies. Blisters, strongly recommended by some physicians, are discountenanced by others, on the ground that they sometimes cause enlargement of the neighbouring glands, which by pressure may in their turn injuriously influence the nerve twigs. Local hot fomentations and the application of leeches are very useful remedies at an early part of the disease, the employment of the latter being generally limited to persons of full habit, and otherwise in the enjoyment of fair health. The endermical application of strychnine—over a blistered surface—the use of various stimulating liniments, and particularly, in the writer's opinion, galvanism, are the more approved remedies in cases which have lasted for a little time.

Before concluding our notice of facial palsy, we must add a few remarks on the occasional occurrence of the disease on both sides of the face, and very briefly refer to the statements of Dr. Todd respecting the integrity of the seventh pair in cases of cerebral hemiplegia, a view which has recently been ably controverted by Dr. Sanders.

Double Facial Paralysis.—This is unquestionably a rare affection, and especially rare when the double palsy is solely dependent on nerve disease. Romberg and Dr. Christison[1] refer to cases of what may be styled simple bilateral paralysis of the face, while the seventeenth case in Dr. Todd's lectures is a very remarkable example of paralysis of the portio dura on both sides connected with affection of the portio mollis; for the patient was "perfectly deaf in both ears;" and the loss of function of both branches of the seventh pair evidently resulted from disease in the temporal bone. In addition to the writers already named, M. Davaine has especially directed attention to the subject in a valuable memoir, the title of which is given below,[2] and to which Professor Gairdner,[3] of Glasgow, in giving an account of a very interesting case of double facial palsy, has referred. Dr. Gairdner considered the paralysis to be due to cold, and connected with rheumatism of the external branches alone; and in the course of his paper he alludes to another case of double paralysis of the portio dura, evidently connected with syphilis. In the latter case iodide of potassium, with iodide of mercury and corrosive sublimate, were employed in alternate doses, and the result was an excellent recovery. One example of double facial palsy has occurred under the writer's observation; it was associated with tubercular disease within the chest, and the patient, a man of thirty years of age, subsequently died of what appeared to be strumous meningitis. Unfortunately an examination of the body after death was not permitted. This is scarcely the opportunity for entering on a consideration of the view which was so strongly entertained and expressed by the late Dr. Todd, that the seventh nerve was very rarely involved in facial palsy depending on cerebral disease, and that the affected facial muscles were those governed by the fifth pair. It will, however, tend to complete the brief exposition of facial paralysis now given, if we state in this connexion, that there is, in our opinion, no reason to doubt that the view taken by Dr. Todd, and in which several systematic writers in this country have closely followed him, is erroneous, and that, on the other hand, the current doctrine on the Continent, and which has been recently ably unfolded and extended by Dr. Sanders, is correct; viz. "that in cerebral hemiplegia, as in peripheral face-palsy, it is the motor seventh nerve which is affected."[1]

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[1] Monthly Journal of Medical Science, 1850.

[2] Mémoire sur la Paralysie générale ou partielle des deux Nerfs de la septième paire: lu à la Société de Biologie (Mars, 1852) par M. C. Davaine. See also Gazette Médicale de Paris, 1852.

[3] Clinical Observations, Lancet, May 18, 1861.

Disease of other of the motor cerebral nerves than the portio dura may likewise determine local palsies. A short reference to such may be made here.

Paralysis due to disease of the third pair of nerves (oculo-motor).—Ptosis or blepharoplegia, the falling down of the upper eyelid, is the notable feature of this affection. When this is due to a cause seated within the cranium, such as an inflammatory exudation, or a tumour, it is almost invariably accompanied by palsy of those muscles of the eyeball, and those fibres of the iris which are likewise governed by the motor oculi. Hence in such cases, and they are far from being uncommon, external squint and dilatation of the pupil are associated with the ptosis. Not only so, but other adjacent cerebral nerves are for the most part implicated, while the indications of the existence of some formidable cerebral lesion are under such circumstances not likely to be absent. On the other hand, when the determining cause of the local paralysis is peripheral in its seat, the ptosis exists alone. Romberg remarks that rheumatism may be the cause of paralysing the palpebral branch of the motor oculi, although not so frequently as is the case with the facial nerve; and he distinctly states that when so induced, the ptosis occurs without the participation of the muscles of the eyeball, and the contractile fibres of the iris.[2] The writer remembers to have seen this dependence of ptosis on rheumatism illustrated in the case of a young lady, who, after having frequently suffered from distinct rheumatic affections, became within a limited period the subject of facial palsy and ptosis, the immediate peripheral impression on both the seventh and third nerves being evidently due to severe cold. A complete and speedy recovery occurred after the local application of warmth and the use of anti-rheumatic remedies. M. Marchal de Calvi has directed attention to the occurrence of oculo-motor paralysis, consequent on very severe tic of the face. M. Marchal, and likewise the late M. Jobert de Lamballe, found the muscles of the eyeball affected as well as dilatation of the pupil, the vision[1] disordered, and insensibility of the conjunctiva in this affection. Such cases, however, are rather illustrative of the reflex form of paralysis, our knowledge of which has been of late greatly increased by the observations of M. Brown-Séquard and others.

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[1] On Facial Hemiplegia and Paralysis of the Facial Nerve, by Wm. R. Sanders, M.D. Lancet, 1865. See on the same subject Dr. Hughlings Jackson in Clinical Lectures and Reports of the London Hospital, 1864.

[2] "Der rheumatische Anlass paralysirt, obgleich nicht in solcher Frequenz wie den Facialis, den Ramus palpebralis des Oculomotorius und hat eine einfache Blepharoplegie ohne Theilnahme der Augenmuskeln und der contractilen Irisfasern, nach der Norm der isolirten Leitung, zur Folge." (Augenmuskellähmung.)

In the same way as peripheral affection of the oculo-motor nerve exists, so may local paralysis result from disease of the *fourth pair* (*trochlear*), and of the *sixth pair* (*abducens*). Such are, however, much less frequent in their occurrence, and especially so, as Romberg has observed, that resulting from affection of the abducens. The author just named has made reference to a case seen by Dr. Dahling, and published by Stromeyer, in which the facial and abducens nerves on the left side were paralysed in consequence of a sudden cooling of the heated face.

Palsy of the tongue from affection of the hypoglossus nerve in its distribution is of great rarity, offering a marked contrast to the frequency with which a central lesion gives rise to the same form of local palsy.

The lesser branch of the fifth pair may be the seat of disease, and consequently give rise to masticatory palsy. The movements of the face in mastication on one or on both sides, as the case may be, are thus arrested or impeded. The temporal and masseter muscles are readily recognised to be inactive; and their condition when the disease is unilateral offers to the touch a marked contrast with the firmness of the same muscles on the unaffected side during the process of mastication. This variety of local palsy, when due to disease of the nerve, is generally caused by tumour of the dura mater, or disease of the sphenoid bone, or such a morbid condition of the gasserian ganglion as compresses the nerve itself.

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[1] Mémoire sur la Paralysie de la troisième paire consécutive à la Névrose de la cinquième. (Archives Générales de Médecine, Juillet 1846.)

LOCAL SPASMS.

By J. Warburton Begbie, M.D., F.R.C.P.E.

The term *Spasm* (*spasmus*, from σπάω, I draw) is used to indicate the sudden and involuntary contraction of muscular fibres or of muscles. *Hypercinesis* (ὑπέρ, in excess, κίνησις, motion) is likewise employed in a sense precisely similar. This peculiar vital phenomenon may be general or local, involving apparently all, or nearly all, the muscles of the body, or, on the other hand, limited to a few muscles,—it may be, to one.

In every occurrence of Spasm there is increased action of the motor nerve, the result of which is the sudden contraction of muscular fibres, the act itself being wholly removed from the control of the will. The expressions *clonic* and *tonic* are used, the former to denote a Spasm which is characterised by rapidly alternating contraction and relaxation of muscular fibres, while the latter implies the existence of the contractions for a certain time, and of this condition rigidity of the affected muscles is also an invariable feature.

Attention is now to be directed to *local* as distinguished from *general* or universal spasms. To the latter, the term *convulsions* is correctly applied.

Local Spasm is not necessarily attended by pain, but it generally is so, and as expressive of painful Spasm we find a suitable term in *cramp* (Saxon *kramp*). The term *cramp* is most frequently applied to painful muscular contraction in the extremities, and to the same phenomenon affecting the stomach or intestines, and also the heart. Such pain as occurs in connexion with Local Spasm is in all probability due to injury done to the sensory nerves supplying the muscle during its violent contraction.

Both kinds of muscular fibre, both orders of muscles, the voluntary and involuntary, are liable to be affected by Spasm. Of the former the most familiar illustration is cramp in the extremities. Of the latter are cardiac and intestinal Spasms. Romberg has pointed out that, as a general rule, when the muscles of animal life, those under the control of the cerebro-spinal nerves, are affected by Spasm, the fibres exhibit a uniform contraction throughout their whole extent; while, on the other hand, the muscles of organic life, over which the sympathetic system is dominant, when similarly affected manifest successive contractions moving like waves.[1]

It need scarcely be observed that, although the abnormal condition now described as Spasm is evidenced by a disorder of muscular fibres or muscles, the cause of this disturbance is always resident in the nervous system. There is a very important and interesting variety in the connexion which subsists between the nervous stimulus and the phenomenon of muscular contraction. The former may be central, that is, operating directly on the great nervous centres, the brain, or spinal cord; or, and in the case of Local Spasm this is far the more frequent, the irritation is peripheral, and consequently the induced action is reflex.

Our knowledge of the causes of Local Spasms is as yet far from being perfect, and in not a few instances the attempt to determine these, notwithstanding the most careful inquiry, signally fails. The etiology of general convulsive disorders is indeed more advanced, and may serve to elucidate doubtful points in relation to the more limited and less serious affection.

The late Dr. Graves of Dublin was one of the earliest to direct attention to the frequency with which various nervous affections, of which Spasm is one, and not the least interesting, are dependent on reflected nervous irritation. He has graphically described the sudden and complete relief afforded to a young lady, who had suffered most severely from spasmodic cough, after the discharge of a tapeworm, which had been effected by a large dose of oil of turpentine with castor oil.[2] The subject thus adverted to by Graves has more recently attracted the attention of several competent observers, more especially of M. Davaine in France,[3] and Dr. Heslop[4] of Birmingham. Their statements show that the presence of worms in the intestinal canal is a frequent cause of remote nervous phenomena, including Spasms, and throw doubt on the assertion of Romberg, that the influence of worms in producing convulsions has been formerly over-estimated. Again, a careful study of the whole phenomena in that most interesting disease, *spasmodic asthma*, has led to the conclusion that the spasmodic affection in it, seated in the smaller bronchial tubes, may be induced by an irritation of the nervous system, which is either centric or eccentric.

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[1] Romberg, Lehrbuch der Nervenkrankheiten des Menschen: Hypercineses, Krämpfe.

[2] Clinical Lectures, Lecture xl., Bronchitic Asthma, Cough.

[3] Traité des Entozoaires. Paris, 1860. M. Davaine remarks:—"Tous les organes, pour ainsi dire, peuvent ressentir l'influence sympathique des vers du canal intestinal: la fausse perception des odeurs, la dilatation de la pupille, l'amaurose permanente ou passagère, l'exaltation de l'ouïe, la perversion du goût, le prurit et les fourmillements à la peau, témoignent de l'action sympathique des vers sur les sens; d'un autre côté, la somnolence ou les vertiges, les rêves fâcheux, *les spasmes*, les douleurs vagues, la toux, la dyspnée, les palpitations, les intermittences du pouls, la faim insatiable ou l'anorexie, la salivation, la qualité des urines, l'amaigrissement, témoignent également de leur action sur le système nerveux, sur les organes de la respiration, de la circulation, de la digestion, sur les sécrétions, enfin sur la nutrition."—Page 48.

[4] The Cerebro-spinal Symptomatology of Worms, especially Tape-worms: Dublin Quarterly Journal of Medical Science, vol. xxvii. 1859.

In the former case the irritation is in the nervous centres themselves, the brain, or spinal cord. In the latter, and it is by far the more common in its occurrence, the irritation is applied at a distance from the nervous centres. This subject has been very fully and ably illustrated by Dr. Hyde Salter, in whose work examples the most interesting and conclusive as to the essentially nervous origin of asthma are to be found.[1]

In treating of what may be styled central asthma, Dr. Salter gave, among others, the following case:—A man about fifty was subject to epilepsy. His fits had certain well-known premonitory symptoms, and occurred with tolerable regularity about once a fortnight. On one occasion his medical attendant was sent for in haste, and found him suffering from violent asthma. The account given by his friends was, that at the usual time at which he expected the fit he had experienced the accustomed premonitory symptoms, but instead of their being followed as usual by the convulsions, this violent dyspnœa had come on. Within a few hours the dyspnœa went off, and left him as well as usual. At the expiration of the accustomed interval after this attack, the usual premonitory symptoms and the usual epileptic fit occurred. On several occasions this was repeated, the epileptic seizure being as it were supplanted by the asthmatic. Nothing seemed to be amiss with the lungs either before or after the attack. Dr. Salter truly observed, that such a case as this appears to admit of only one interpretation, that the particular state of the nervous centres that ordinarily threw the patient at certain times into the epileptic condition, on certain other occasions, from some unknown cause, gave rise to bronchial Spasm; that the essential diseased condition was one and the same, but that its manifestation was altered, temporary exaltation and perversion of the innervation of the lungs in the asthmatic paroxysm supplanting unconsciousness and clonic convulsion in the epileptic seizure. It has occurred to the writer to witness in one instance an alternation of phenomena bearing a close resemblance to that observed by Dr. Salter. The patient, a young man, was admitted to the Royal Infirmary of Edinburgh, on the recommendation of Dr. Turner of Keith. He had for several months previously been subject to cerebral attacks, attended by loss of consciousness, and occasionally by convulsive movements of the muscles of the face and extremities. These continued to occur during the patient's residence in the hospital, observing for a time the same periodicity which had antecedent to that time always distinguished them, when, on three separate occasions, and in the most distinct manner, an attack of asthma took the place of the more manifest cerebral disorder. The loss of consciousness and convulsive movements again recurred in a modified form: and after the lapse of several weeks, during which various remedies were employed, the patient left the Infirmary to return home, his condition having materially improved. Besides instances of the nature just alluded to, there are other examples of asthma, which, although in by no means so distinct a manner, must be held as caused by some impression taking origin in the nervous centres, and responding in a mysterious manner with certain feelings or emotions of the mind; such are the cases in which fear, excitement, and fatigue operate.

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[1] On Asthma: its Pathology and Treatment. London, 1860. See also article on Asthma, Vol. III. p. 512.

Now, passing to a very brief consideration of *bronchial* Spasm, dependent not on centric but *peripheral* irritation. Dr. Salter speaks of three degrees of remoteness of the application of the stimulus producing asthma, and consequently of three groups into which the reflex cases of the disease may be divided :—1st. Those in which the source of irritation is alimentary, and chiefly gastric. 2d. In which the irritation is more remote, but still confined to the organic system of nerves; as, for example, asthma produced by a loaded rectum, by the presence of tape-worm, or ascarides. 3d. Cases in which the cerebro-spinal system is the recipient of whatever irritation is the cause provocative of the attack, as, for example, was illustrated in a most remarkable instance recorded by Dr. Chowne, where the application of cold to the instep produced in the most direct manner the asthmatic paroxysm. Looking to the first, and by a long way the largest, of these three classes of cases, the nerve irritated is the gastric portion of the pneumogastric; through it the stimulus reaches the medulla oblongata, and from that portion of the nervous centre it is again transmitted to the bronchiæ by the pulmonary filaments of the same nerve. It is indeed of the highest importance in a therapeutical point of view to notice this chain of connexion. We are thus called to recognise in the paroxysm of asthma a disease not unfrequently originating in disorder of the stomach; and it may be assumed, as a correct conclusion, that a large proportion of the sufferers from this severe spasmodic affection are to be relieved by attention being given to their diet and regimen. But even here we should be adopting too limited a notion of the influence of the digestive and assimilating processes in the production of asthma, did we conclude that those cases alone are examples of this nature, in which bronchial spasm is induced by reflex stimulation directly through the important nervous trunk—the pneumogastric. There are, over and above, numerous instances in which this direct communication of the influence exerted will not apply. In such the occurrence of the Local Spasm does not so speedily follow the introduction of food into the stomach as in many of the former cases, and therefore we must look for a somewhat different explanation. We find it in the disordered condition of the blood; the faulty assimilation is no doubt the primary cause of this, but the unhealthy blood is in such instances the direct irritant; by its operation on the nervous distribution through the lungs the bronchial spasm is caused. This humoral origin of asthma affords in all probability the most satisfactory explanation of the frequent occurrence of this nervous disorder in persons who are gouty. The accuracy of the view thus expressed is further evidenced by the circumstance that such sufferers are benefited by a plan of treatment which tends to eliminate the essential poison of gout from the system; often, indeed, are benefited by such a plan of treatment only. In these cases remedies need scarcely be directed to the chest: it may be possible to relieve, it is impossible to subdue, by antispasmodics a bronchial spasm so induced; but on the other hand, by acting freely on the great emunctories of the body, on the skin and kidneys, the disease is to be met and overcome.[1]

Allusion has been made to the production of bronchial Spasm as determined by reflex irritation, and also by an impure condition of the blood. The same precisely holds true of *cardiac* Spasm. The irregular, unrhythmical, and painful contractions of the heart known under the name of *palpitation,* are found in close connexion with various derangements of the general health, and of special organs. Among the latter, those of the alimentary canal, but particularly of the stomach, and of the uterus, occupy the chief place. Perhaps the most painful of all the forms of cardiac palpitation is that resulting from either an imperfect depuration of the blood, or from a regular blood impoverishment, or anæmia, as is so frequently observed in cases of amenorrhœa and chlorosis.

We pass to a brief consideration of Spasm as occurring in the muscular organs which constitute the alimentary canal. It affects *the stomach and intestines* as well as the *œsophagus and pharynx,* while the severe pain determined by its occurrence in any part of the alimentary tract is very generally accompanied by other and various symptoms which cannot with any propriety be referred to now. Painful peristaltic spasm of the intestines is usually known under the name of *colic.* During its occurrence, and as affording proof of its occasional violence, *intussusceptio,* and prolapsus of the rectum may take place. Foremost among the determining causes of colic is to be placed the presence of indigestible articles of food and morbidly altered secretions in the intestinal canal. But, besides this, the influence of the emotions, and more especially of fear and fright, is well known; just as bronchial Spasm may be due to reflex nervous irritation, so may intestinal-spasmodic stricture (as it is called, to distinguish a temporary and functional from an enduring and organic contraction, similarly produced) have its seat in any part of the alimentary canal. In some instances the direct exciting cause is seated at a great distance from the induced disorder: of this nature no more common or manifest example can be given than that of colic, often very severe, resulting from the exposure of the lower extremities, it may be of the feet only, to cold and damp. *Spasm of the pharynx and œsophagus* is one of the most interesting of all the varieties of Local Spasms. It is of common occurrence, particularly in females, in whom it shows itself either as a reflex phenomenon dependent on uterine irritation, or—and this still more frequently—as one of the most striking features in a paroxysm of hysteria. It is not always an easy task to distinguish between spasm of the œsophagus due to organic disease and that which is simply the result of a nervous irritation. The cautious introduction of the probang or œsophageal bougie is the most ready and certain means for establishing the diagnosis.

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[1] Laennec, who, while strongly insisting on the connexion between asthma (asthme spasmodique) and catarrh, admitted the existence of a purely nervous asthma (sans aucune complication de catarrhe), has acknowledged the great difficulty there is in the satisfactory treatment of the disease. "Beaucoup de moyens," he remarks, "peuvent être opposés aux troubles de l'influence nerveuse qui constituent principalement l'asthme: *mais ici, comme dans toutes les affections nerveuses,* rien n'est si variable que l'action des médicaments: les remèdes qui réussissent le mieux chez un grand nombre de sujets sont sans efficacité pour beaucoup d'autres; et chez le même individu tel moyen qui avait produit d'abord des effets héroïques, et d'une promptitude surprenante, devient tout à fait inefficace au bout d'un petit nombre de jours. Il faut successivement en essayer plusieurs, et souvent de très-disparates: nous allons, en conséquence, parcourir les diverses séries de moyens dont on a tiré le plus d'avantage dans l'asthme."—*Traité de l'Auscultation Médiate: Affections Nerveuses du Poumon.*

An irritation of the pharynx or œsophagus, of the stomach, bowels, or liver, is sometimes the direct cause of *hiccup* or *singultus*, a spasmodic affection extremely interesting in its nature. Sudden powerful jerking inspirations, accompanied by a peculiar noise, and succeeded by a brief expiration, interrupting speech, distinguish hiccup. It is essentially a reflex phenomenon; in the vast majority of instances depending on some peripheral irritation, but occasionally as its presence in apoplexy, meningitis, and hydrocephalus testifies, determined by a central cause. There seems to be some difficulty in accounting for the occurrence of hiccup from an irritation of the phrenic nerve, as has been suggested by various writers; nevertheless it is consistent with the writer's observation in several instances of long-continued and distressing hiccup, that firm pressure exerted for a brief period over the lower part of the neck, corresponding to the situation of the scaleni muscles, so as to probably compress the phrenic, has led to its temporary and even entire arrestment. In singultus and in yawning, which resembles it in being of the nature of inspiratory convulsion,—also in *sternutatio* or *sneezing*, where the expiratory function is involved,—what is of consequence to notice is, as Romberg has pointed out, that the spasmodic action does not affect a single muscle, but, on the contrary, groups of muscles; and that these Local Spasms, more particularly the former, hiccup, while occurring as independent affections, are still more prone to assume the symptomatic character, affording evidence of the existence of some other malady, or distant irritation.[1]

Spasms of the *urinary bladder* and of the *urethra*—the latter commonly styled spasmodic stricture—are familiar to the surgeon. Vesical Spasm is not unfrequently a truly reflex phenomenon: this is witnessed on the introduction of the catheter or bougie, when violent and most painful efforts are made to evacuate the organ, even when at the time empty. Romberg insists on the action of the vesical muscles being due to an irritation of the neck of the bladder, that particular part being, as Sir Charles Bell demonstrated, the most vascular and the most sensitive portion of the viscus. It is when the catheter reaches, or the calculus touches, the neck of the bladder, that the ischuria is produced; and the intense pain is seen to subside whenever the irritating body is removed from that particular portion of the organ. The irritation upon which vesical spasm depends may, as we have seen to hold true of other forms of Local Spasm, be distant from the induced phenomenon. It may be resident in the kidneys, or in any part of the intestinal canal, but very specially in the rectum. Hæmorrhoids are a frequent cause of vesical spasm; and it is well known in how distressingly severe a degree that is apt to occur after the operation of their deligation. Exposure of the surface of the body, especially of the feet, to cold and wet, and depressing mental emotions, act in the same way.

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[1] "Häufiger als auf einzelne Nervenbahnen beschränkt, kommen die krampfhaften Athembewegungen zu Gruppen associirt vor, entweder selbständig, oder was öfter der Fall ist, abhängig, und in Begleitung von andern Affectionen." (Loc. cit. p. 354.)

As our object in this article has been, not to illustrate every example of Local Spasm, but rather to indicate the nature of this special morbid action by a brief consideration of some of its more important and most frequently occurring varieties, we shall now take a very rapid survey of a few other forms, and bring our remarks to a conclusion by offering some general observations with a special reference to treatment. There is a peculiar variety of Local Spasm affecting certain muscles of the face, and giving while it lasts a very strange aspect to the individual. In the *histrionic spasm of the face*, by which title this affection is known, there are, in the language of Romberg, "grimaces, alternating or lasting, on one side, seldom on both sides, of the face."[1] Pain is occasionally, but by no means necessarily, an accompaniment of the disordered muscular action. A local malady essentially, because affecting the muscles governed by *one* nerve, the seat of the spasm is in some instances still further localized by there being only one of the branches of the seventh pair involved. Of the latter are *blepharospasmus*, or spasm of the eyelids, and the *risus caninus*. The peculiar convulsive grin thus named is caused when the molar and labial branches are affected. To it the terms *spasmus cynicus* and *sardonic laugh* are likewise applied. The relation of facial spasm to chorea must not be overlooked; this association has been frequently noticed: and it is also a matter of not unfrequent observation that the Local Spasm lasts in some cases for a considerable period after the disappearance of the general nervous disorder with which it had been in the first instance connected.

Masticatory Spasm is witnessed in its most formidable degree when, as trismus, it accompanies, or is itself the chief element in, tetanic convulsions. In a much milder degree spasm in the muscles which are supplied by the motor division of the fifth pair is seen as a reflex action, determined, as in children, by the presence of worms in the intestinal canal, or by the progress of dentition. The spasm of the muscles is sometimes associated with a grinding of the teeth. To the occurrence of the latter symptom in persons of the gouty diathesis attention was called by the late Dr. Graves. Such grinding of the teeth continued for years as a daily habit, and produced very remarkable changes in the conformation of these organs, affecting sometimes one side of the jaw, sometimes both; so that in confirmed cases the teeth were frequently found ground down to the level of the gums.[1]

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[1] Loc. cit. "Mimischer Gesichtskrampf."

Spasm of the muscles of the eye, dependent on an irritation of the third, fourth, or sixth nerves, is seen in *strabismus*,—which is to be distinguished from the paralytic form by the movement of the eyeball in other directions being in the former case possible,—and in *nystagmus*. These spasmodic affections equally with others acknowledge a peripheral or central origin. Both are of common occurrence in connexion with intestinal and dental disorders, but they are also not unfrequently the indications, sometimes among the very earliest, of mischief, inflammatory or otherwise, commencing at the base of the brain.

Painful Spasms of the muscles of the extremities are of very frequent occurrence; and with this affection, more especially seated in the lower limbs, and then in the calves, we are especially familiar under the name of *cramp*. The attack of cramp is usually sudden; and it frequently occurs at night, the person in bed being awakened from sleep by the seizure. During its continuance the muscular fibres are gathered up into a hard knot, which is always easily felt by touch, and may often be seen. The pain is very severe, and produces a feeling of sickness and depression, which may even lead to syncope. The patient not unfrequently gives utterance to an irrepressible exclamation or scream. Cramp usually lasts only for a few moments; it may, however, continue for minutes, and even hours. A sudden cessation of the spasm may occur, or a more gradual relaxation of the muscular fibres ensue; but in either case, if the attack have been at all severe, sufficient injury during its continuance has resulted to the sensory nervous filaments as to cause a feeling of soreness, always increased by touch, and frequently an inability fully to exert the affected limb or other parts for some time. The irritation of the sciatic nerve, upon which the painful spasm of the muscles of the calf depends, is intimately connected with disorder of the stomach and bowels, and is also particularly prone to occur in persons of the gouty and rheumatic habits.

In Asiatic cholera the occurrence of intensely painful cramps contributes, as is well known, largely to the sufferings of its victims. Again, in persons of intemperate habits there is sometimes observed a tendency to the development of severe spasmodic action in the muscles, of the extremities more especially, but likewise of other parts of the body. In one instance which fell under the writer's observation, a patient, having recently recovered from an attack of delirium tremens, was seized with most violent and painful spasm of the muscles of both upper and lower extremities, during which the fingers were powerfully flexed and bent inwards on the palms of the hands, as in the carpal contractions of children. So severe was this case, that a syncopal depression, very threatening in its character, occurred. After lasting for several hours, and exhibiting for many days a marked tendency to recur, the affection passed off, and the patient entirely recovered both health and strength.

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[1] Clin. Med., "Gout."

In the *treatment* of local, as of general, Spasms, the great object is to remove the cause on which they depend. In the brief consideration of the different varieties of Local Spasm now offered it has been shown that in a large proportion of cases the excited muscular action is induced by reflex action; that the direct exciting cause is a distant nervous irritation. Fortunately the removal, or at all events the lessening, of this irritation is in many instances within the power of our art. Again, in those cases, of the frequent occurrence of which proof has been afforded, which are characterised by a morbid state of the blood, *e.g.* gouty or rheumatic, we may often be successful in our treatment by paying due attention to the therapeutical indications,—in other words, by the employment of an alterative or eliminating plan, suggested by the peculiarity of each individual case. We may as effectually subdue the morbid action of Spasm as we are constantly enabled, by the use of suitable remedies, to relieve that of pain in neuralgia. In addition, we possess in various agents a power of controlling or completely removing such excited nervous action as induces Local Spasm: not indeed one upon which we can invariably rely, because we are often disappointed in the results; nevertheless the remarkable therapeutical effects which succeed the exhibition of various of the antispasmodic and calmative remedies is such as to convince us of their efficiency. Our knowledge, moreover, regarding the action of such remedies is on the increase. It is only quite recently that a valuable addition has been made in the bromide of potassium, the operation of which in removing the painful cramps of cholera, not less than in many instances averting the convulsive seizure of epilepsy, has been witnessed by numerous observers.[1]

Pressure firmly exerted on the thigh relieves a violent cramp of the calf, while, according to Dr. Wise, the application of a tourniquet so as to compress the blood-vessels will banish the exhausting muscular contractions in cholera.

Finally, in the treatment of such exalted nervous action as determines Local Spasm, as in the proper management of every form of derangement of the nervous system, however slight or severe, let the potent influence of peculiarity in psychical constitution, and of the ready susceptibility in some to the operation of all manner of external impressions, not be lost sight of.

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[1] See Note on the Therapeutical Effects of Bromide of Potassium, by James Begbie, M.D.; Edin. Med. Journ. 1866. Also, The Actions of Bromide of Potassium upon the Nervous System, by J. Crichton Browne, M.D.; Ibid. 1865.

TORTICOLLIS.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—A spasmodic condition of the muscles of the neck—generally clonic, but rarely tonic—whereby the head is displaced to one side, or towards one shoulder, or is thrown backwards; occurring almost exclusively in adult life, and characterised by great obstinacy and chronicity.

Synonyms.—Wry-neck; spasmodic wry-neck; spasm in the muscular distribution of nervus accessorius Willisii, and of the superior cervical nerve (Romberg).[1]

Causes.—So far as I have seen, the male sex has been slightly more frequently affected than the female; but the difference is so small, that its existence is of no diagnostic value. The affection has sometimes originated, and recurred, or been exaggerated, during pregnancy. With only few exceptions, the cases that I have seen have first presented symptoms after thirty years of age; and the majority after forty. There has been no one thing, nor any combination of circumstances, which has occurred with such frequency as to warrant a belief in its operation as an *exciting cause.* Once a strained position, maintained for a long time; occasionally exposure to cold; sometimes a sudden shock, either mental, moral, or physical; and at other times the presence of long-continued anxiety, or the recurrence of pregnancy, has been referred to by the patient as the cause of symptoms; but, in regard of such modes of causation, we can see distinctly that which might lead to disturbance of the nervous system of any kind whatsoever, but we fail to see anything which should conduce to this special form of derangement.

In one case that I have seen the symptoms were preceded by hemiplegia; in another by paralysis agitans of the side from which the head was turned; in a third, and fourth, and fifth, there was previous "writer's cramp;" in a sixth there was histrionic spasm of the face; but in the majority of cases the nervous system had exhibited no prior derangement, and had continued free from ulterior disturbance for a long period of years.

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[1] Syd. Soc. Transl. of Manual of Nervous Diseases of Man, vol. i. p. 316.

The position in life and the occupations of those who have suffered from Torticollis have varied widely, and I have not been able to attribute the malady with anything like constancy to that common cause of nervous disease,—overwork.

SYMPTOMS.—There is great similarity in the symptoms presented by different individuals, when once the disease is established, and is free from accidental complications. Sometimes the *commencement* is sudden, but much more commonly it is gradual, and often so insidious at first that the real nature of the malady is overlooked. The patient feels uneasy in the neck, thinks that something is wrong with his cravat, or with his pillow, and only after several months discovers for himself, or is told by others, that his head is not straight. There is with this want of symmetry some uneasiness in the neck, extending from the occipital protuberance downward to one of the shoulders, and sometimes onwards into the arm, or even forearm. As the malady *advances* the uneasiness becomes greater, and sometimes amounts to definite pain, felt usually in the same direction. The pain is increased by voluntary efforts to bring the head into the middle line, but sometimes attains its maximum when the head is carried round to the furthest point possible by the spasmodic movement. The pain is not severe, but generally of dull, aching character; and often is relieved by lying down, and keeping the head still by resting it upon a pillow.

Observed casually, a case of medium severity would give the impression to a bystander that the patient's cravat was uncomfortable, and that he was trying to make it less so by moving the head, in a somewhat restless manner, towards one side; or that he was making some attempt to look at an object on one side of him, which object he could not "get his head round" sufficiently far to see conveniently.

Upon more careful examination it is seen that the head is constantly being moved, by a succession of jerks, in such manner that the occiput is depressed, and the chin raised, and that the movement is in a definite direction, hour after hour, and month after month. Early in the case the individual is able so far to antagonize the spasm, by a simple voluntary effort, as to bring the head into the middle line, or even beyond it; but as time passes on this often becomes impossible, and the hands are used to pull the head back again into its proper position.

When Torticollis has existed for a few months only, the head presents a constant series of movements,—the spasm and the voluntary effort so balancing one another that the effect is that described above. But when it has lasted for a longer period, the head is habitually "carried on one side;" for the voluntary interference with the spasm, although frequent,—if not constant,—does not suffice to bring the head into a central position, being overcome by the spasmodic contraction. Sometimes, even under these circumstances, a very strong voluntary effort may restore momentary equilibrium; but the effort is attended by distress, if not by pain, and is often followed by an exaggeration of the spasm.

The muscles of the neck on the side from which the chin is turned are found hard, contracted, and often hypertrophied; those on the opposite side are frequently soft, and sometimes wasted. Early in the history of Torticollis it would seem—so far as my experience extends—that the deeply-seated muscles at the back of the neck are the most affected; the sterno-mastoid, at such time, being often free from spasm. At a later period the sterno-mastoid is found hard, frequently hypertrophous.

Occasionally the muscles of the shoulder are so involved that the acromion is raised; more rarely the muscles of the face present histrionic spasm; and not unfrequently there is some difficulty in controlling the movements of the arm. I have noticed sometimes difficulty of deglutition, and in a very few cases some morbid condition of motility in the leg: but these symptoms must be regarded as complications rather than conditions of the disease now under consideration; for it more frequently happens that the muscles of the neck are alone involved in the morbid contraction.

As a rule, to which the exceptions are very rare, the spasms cease during sleep; and not only so, but when the patient lies down and supports the head. They are increased by all that lowers or disturbs the general health, and by emotional excitement.

The electric irritability of the contracting muscles I have found much increased when tested by faradisation: the electric sensibility is sometimes so greatly augmented that an interrupted current, not in the least degree painful on the healthy side, was perfectly intolerable when passed through the seat of spasm. It has appeared often that the relaxed muscles, on the side opposite to the contraction, have exhibited less than their normal contractility; but I have never found them so defective that it was impossible to restore the head to equilibrium by their direct faradisation. The battery current, when continuous, and passed through the contracting muscles, relaxes the spasm and allows of temporary equilibrium; but, when interrupted, its action is similar in kind to that exerted by the induced current of faradisation: there is, however, less intensity of contraction, and much less display of electric sensibility. The effects of either the battery current or of faradisation appear to be singularly transient, in whatever manner they may have been produced. It has often happened to me to see that a head which had been maintained *in equilibrio* for many minutes, and that day after day for a considerable number of days, returned at once to its spasmodic jerkings the moment that the application was suspended. Sometimes it has been obvious that the spasm was subsequently increased by the electricity.

The side to which the twisting occurs has been sometimes the right, sometimes the left. There appears to be no special proclivity to the affection of one side rather than the other in either sex; but when once the malady has shown itself, its pertinacity is remarkable: it remains in exactly the same position, with slight tendency to extend; or it may in rare instances disappear for eight or nine years, and then return to the muscles that it had previously affected. In many cases *progress* is so slow that no change is observable after several years—*i.e.* no change as to locality—whereas in others the malady seems to extend either upwards or downwards, and involve muscles not implicated at the first. In this manner the face may be distorted or the arm may be rendered partially useless by either rigidity or weakness; the head becomes more or less fixed in an oblique position, the ear of one side being drawn down to the shoulder, and the chin thrown upwards and outwards in the opposite direction. When left entirely to itself,—*i.e.* when not interfered with by either the will, the ideas, or emotions of the patients, or by any influence from without,—the spasm is tonic, and the head may remain for hours drawn to one side, but motionless. This is rarely, if ever, noticed early in the history of a case, and sometimes it is never observed; but, even when it occurs after several years' duration of the spasm, the slightest emotional disturbance or attempt at voluntary movement brings back the clonic contraction: and the only difference to be recognised between the early and the later stages of the malady is, that in the latter the head is never brought back to the position of exact equilibrium, and that there is less obvious movement of the head; for, as it seems, the habitual struggle between volition and clonic spasm is given up, and the latter, having gained the day, allows tonic spasm to take its place.

The mental faculties, the sensibility of the skin, the special senses, and the general health undergo no necessary changes in Torticollis, but I have often observed great mental depression. In some highly-marked instances there has been complete integrity of function in every direction; the one thing that has been wrong has been the disease itself. Sometimes the general health has been impaired, the patient has been anæmic and weak; but this has been frequently the result of the annoyance occasioned by the spasm, and very rarely the supposed cause of its development.

Numbness and anæsthesia may occur in the arm, together with œdema, when the scaleni are so much affected as to press upon the brachial plexus and its adjacent veins.[1] Insomnia is by no means unfrequent.

DIAGNOSIS.—The symptoms that have been now described are sufficient when carefully regarded to enable the practitioner to distinguish this disease from every other. An accidental exposure to cold may produce "stiff-neck;" but here the head is permanently fixed in one position, and maintained therein, not by spasmodic rigidity of muscle, but by the fear of pain which, as the patient knows, any movement may occasion. Such malady has its relations to pleurodynia, lumbago, and "muscular rheumatism;" it is sudden in its development, and temporary in its duration, and could only be accidentally mistaken for Torticollis. The opposite error is sometimes made,—viz. that of regarding genuine spasmodic Torticollis as a simple "stiff-neck from rheumatism or cold." In its earliest stage, however, genuine Torticollis should be at once distinguished by the clonic character of the spasm, and freedom from pain on movement.

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[1] Romberg, loc. cit. p. 317.

Injuries to the spine occasionally produce stiffness of the neck, and this to such a degree that the head may be permanently placed in some awkward position. In such cases the spasm is tonic; there is marked tenderness of the spinous processes, and with this some fulness or hardness around or behind the vertebral column; and there is also some impairment of the motor or sensory properties in the arms and legs.

In certain *organic diseases of the brain* accompanied by hemiplegia there is sometimes Torticollis, just as there is synergic movement of the eyeballs; but the mode of onset of symptoms is such that a case of cerebral apoplexy cannot well be confounded with the malady now under consideration. The opposite mistake has, however, sometimes been made, and an individual who is beginning to suffer from Torticollis spasmodica has been supposed to be the subject of organic disease of the brain. For the distinction between these two very different conditions it is sufficient to bear in mind that in the one the disease is limited to the neck, in the other it occurs in combination with marked hemiplegia; that in the former the spasm is clonic, in the latter tonic; and that in the first the development of symptoms is insidious, gradual, and local, whereas in the second it is sudden, and of wide distribution.

It is enough to mention the existence of cases in which *growths*, benignant or malignant, may affect the position of the head, in order to prevent the occurrence of any errors in diagnosis.

PATHOLOGY.—Anatomical inquiry has not yet shown the locality or existence of any special lesion of the nervous centres with which Torticollis is necessarily associated. Physiological experiment has proved that it may exist when the spinal accessory nerve is irritated at its passage through the foramen lacerum,[1] or when injury is inflicted on certain muscles, upon the olivary body, or the auditory nerve.[2] The disease would appear to be one of those curious conditions—not yet fully understood—in which some "centre" of associated movements is so altered that there follows a disturbance of the normal equilibrium; a disturbance exhibiting itself at first by dynamic change, but subsequently leading to structural alterations in the affected muscles. It has its analogies in writer's cramp and histrionic spasm, and its peculiar and intimate pathology is a question as yet reserved for further investigation.

PROGNOSIS.—When once established,—*i.e.* when fully developed and of three or four months' duration,—Torticollis is one of the most obstinate of maladies. It has sometimes yielded to treatment, under favourable circumstances; but it has almost invariably recurred, and has proved capable of resisting all efforts made for its relief.

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[1] Volkmann, quoted by Romberg, loc. cit. p. 316.

[2] Brown-Séquard, Lectures, p. 194.

When the case departs widely from the ordinary type,—as, for example, when the chin is drawn either backwards and upwards, or downwards and forwards, by bilateral contraction of the muscles at the back of the neck, or at its front,—the prognosis is more favourable. Such cases frequently improve, and sometimes get well by rest, and other measures.

Unfortunate as the prognosis is with regard to the cure of this special malady, there is one ground for consolation,—viz. that it is not by any means necessary, nor is it at all highly probable, that the victim of Torticollis should suffer from any other nervous disease. Sometimes it forms but part of a general nervous disorder; but, as a rule, it exists alone; and although it may continue for many years, the source of great but measurable annoyance, it does so without entailing any danger to life, or any high probability of ulterior change. Prognosis, therefore, is based upon the duration of the disease, and its complication with other signs of nervous malady. When it exists alone, the patient may look forward to a troublesome and obstinate affection; but he may, at the same time, know its limits, and be directed to go on without fear of further mischief.

TREATMENT.—In its early stages Torticollis has yielded to various plans of treatment; iron, setons, moxæ, rest, mercurials, electricity, pressure on the cervical sympathetic, and the division of nerves or of muscles, have each been followed by a cure: but in the advanced stages no one, nor any combination, of these modes of treatment has availed to cure, or even to modify, the disease.

I have used all kinds of soothing applications, have employed electricity in every form, and have failed to influence the disease when once it has become fairly fixed; but have found that the continuous current has been useful when the malady has existed for a few months only, and have also at that period seen notable advantage from the continued application of morphia by the method of hypodermic injection.

It would seem desirable to enjoin rest; to secure the regulation of the general health; to apply a moderate continuous current to the muscles which exhibit spasm, and a mild induced current to their antagonists; and to inject morphia, hypodermically, for a lengthened period. It is not essential that the morphia should be injected into the neck; it may be introduced into the arm or thigh, or any other convenient locality: but it is important that its use be steadily continued, and that the quantity injected be gradually increased until a definite effect is produced upon the spasm. Beginning with the tenth part of a grain, the quantity may be increased, if necessary, until two, or even three, grains are injected twice daily; and when the patient can bear this amount, the spasm has sometimes yielded. But it often happens that morphia, even by hypodermic administration, cannot be borne, from the fact of its producing nausea, constipation, and an amount of malaise that is greater than the evil it is intended to relieve ; and in such cases the Torticollis is positively increased by the injection. Several patients whom I have known with Torticollis have positively refused to continue the injection of morphia from the misery which it has thus occasioned.

Mechanical contrivances have been employed in order to force the head into position ; but these, although so managed as to be borne for a short time,—*e.g.* to enable a clergyman to get through a service, or a doctor to visit two or three patients in succession,—are often found to be productive of so much annoyance, or even pain, that the patient would rather trust to his own hands or to the "chapter of accidents" in order to get through his work. The most simple, and at the same time most effective, appliance that I have seen for mild cases is that devised by Dr. Hearne of Southampton ; but it has failed to be of service when the disease has been of long duration. Mr. Heather Bigg has constructed several machines which meet the difficulty for short periods of time ; but I have not yet seen any apparatus which a patient with confirmed Torticollis could bear habitually.

Division of the nerves has been useless,[1] and division of the sternomastoid worse than useless, for it has led to an exaggeration of the spasm in the deeper-seated muscles at the back of the neck, as I had occasion to observe in a well-marked case that came under my notice some years ago.

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[1] Romberg, loc. cit. p. 319.

LOCAL ANÆSTHESIÆ.

By J. Warburton Begbie, M.D., F.R.C.P.E.

The term Anæsthesia (*α* privative, *αἴσθησις*, sensibility) indicates deprivation or loss of sensibility, and was first employed by the distinguished Cappadocian physician, Aretæus.[1] There exist three abnormal modifications of the function of sensation: *first,* it may be lost; *second,* it may be exalted (hyperæsthesia); *third,* it may be perverted.

By Local Anæsthesia we understand a morbid state of sensibility, in which the normal physiological sensation of a part is abolished entirely, or nearly so.

Since the introduction of ether and chloroform inhalation, for the purpose of destroying pain, it has been customary to describe these valuable agents as anæsthetics, and the condition of insensibility into which the person is thrown by their action as Anæsthesia. With this interesting phenomenon we have at present no concern.

In Local Anæsthesia the want or failure of the due impression must arise from a morbid state of the extremities of nerves, or of an afferent nerve ceasing to convey the impression to the sensorium, or of the sensorium itself.

Thus we are entitled to limit the seat of the morbid influence, because these three organs, or classes of organs, are concerned in the production of each sensation.

With precisely the same signification as Anæsthesia, the expression paralysis of sensation, or of the nerves of sensation, has been employed. It were better, however, to abandon the use of paralysis in this sense altogether, and to restrict it to the loss of power of motion. The intimate connexion of paralysis and Anæsthesia is abundantly conspicuous: the latter is very frequently noticed as an antecedent phenomenon of the former, or they occur simultaneously; and while paralysis lasts Anæsthesia may continue, or sensation may be restored long before the recovery of the power of motion.

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[1] ἢν δὲ ἁφὴ ἐκλείπη μούνη κοτέ,—σπάνιον δὲ τὸ τοιόνδε,—ἀναισθησίη μᾶλλον ἢ πάρεσις κικλήσκεται.—Περὶ Παραλύσεως. Περὶ Αἰτιῶν καὶ Σημείων Χρονιῶν Πάθων. Βίβλιον Πρῶτον.

The special situations in which Anæsthesia is met with, or may be considered apt to occur, are various. For convenience of illustration the following classification may be made, and to the forms now to be mentioned attention will be very briefly directed:—(*a*) Anæsthesia of the skin (cutaneous Anæsthesia). (*b*) Anæsthesia of muscular nerves. (*c*) Anæsthesia of sensorial nerves. (*d*) Anæsthesia of the fifth pair of nerves. (*e*) Anæsthesia of mucous surfaces. (*f*) Anæsthesia of the viscera.

(*a*) *Anæsthesia of the Cutaneous Nerves.*—The notable and lasting diminution, or the entire loss, of the tactile sense of the skin is what is understood by cutaneous Anæsthesia. It is by a careful examination as to the delicacy of tactile sensibility, and the perception of degrees of temperature, that we are enabled to determine the extent to which Anæsthesia of the surface exists. For the former purpose the mere statements of the patient will not suffice. Besides measuring the degree and determining the precise seat of Anæsthesia by the point of the needle, recourse must be had to the method of experiment suggested by Weber, testing the consciousness of the patient, while blindfolded, to the two points of a pair of compasses, placed at different parts upon the skin, or, which is still more satisfactory, employing the delicate little instrument known as the æsthesiometer of Dr. Sieveking. The ready and accurate determination by the patient of degrees of temperature, heat and cold, is impaired or destroyed: it is not uncommon to find hot things styled cold, and cold things hot. In marked instances of cutaneous Anæsthesia the power of resisting the injurious influence of temperature is lost; and not only so, but, owing to a similar defect, superficial sores are readily formed on parts of the body exposed to even a slight degree of pressure. Evidence of the derangement of the circulation is afforded by a change in the colour of the affected part; it is apt to become livid or blue in appearance, and extravasations of serum, and even of hæmatin, occur. Distressing sensations are experienced by the patients,—chiefly numbness and pricking; also formication.

In alluding to the treatment of cutaneous Anæsthesia, the distinguished German writer on nervous diseases truly observes, "Die Behandlung der Anæsthesia cutanea war bisher eine *oberflächliche*, im wahren Sinne des Wortes;" but while this is to be regretted, we may reasonably anticipate an increase of our knowledge, owing to the much more satisfactory manner in which the causes and seat of disease have of late, and are at the present time, being investigated.

(*b*) *Anæsthesia of Muscular Nerves.*—The loss of the power of motion is usually unassociated with any marked degree of muscular Anæsthesia. On the other hand, instances are on record in which a very perfect insensibility to pain has existed in muscles, while the power of moving them has been retained. It is of the utmost importance to distinguish between the loss of tactile sensation (cutaneous Anæsthesia) and the definition of sensation in muscles, for without carefulness in examination these two are capable of being, and in some instances have no doubt been, confounded. Romberg makes the interesting observation, that muscular Anæsthesia, without the loss of or any damage done to tactile power, exists in tabes dorsalis.[1]

(*c*) *Anæsthesia of Sensorial Nerves.*—The nerves of special sense which thus suffer are the optic (Anæsthesia optica), the Auditory (Anæsthesia acoustica), the Olfactory (Anæsthesia olfactoria; Anosmia), and the Gustatory (Anæsthesia gustatoria; Ageustia). To the many interesting affections included under these terms—for example, amblyopia and amaurosis under optic Anæsthesia—it is not desirable to make any reference now. Such important diseases demand a separate and detailed consideration not contemplated in this System of Medicine.

(*d*) *Anæsthesia of the Fifth Pair of Nerves* (Facial or Trigeminal Anæsthesia).—Physiological experiments have demonstrated the remarkable effects produced by section of the fifth pair; of these, insensibility of the face, eye, nostrils, cavity of the mouth and tongue, is the most conspicuous: while the extent of the Anæsthesia is of course determined by the nervous injury being limited to one or more branches, or, on the other hand, involving the trunk before division. Experimental inquiry, as well as clinical observation, have further shown that when injury or lesion of the nerve exists within the cranium, the resulting phenomena are not such as are included in Anæsthesia merely, but paralysis and impairment or loss of special sense are also induced. Romberg,[2] in directing attention to the different diagnostic symptoms, has indicated certain very important particulars, as follows:—(*a*) The more the Anæsthesia is confined to single filaments of the fifth pair, the more peripheral the seat of the cause will be found to be. (*b*) If the loss of sensation affects a portion of the facial surface, together with the corresponding facial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium. (*c*) When the entire sensory tract of the fifth nerve has lost its sensation, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease. (*d*) If the Anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain. Thus facial Anæsthesia, as a phenomenon of disease, may be in itself a simple, really trivial, affection, or it may be the indication of serious organic disease. In the former case it will be apparently independent and isolated; in the latter, linked with other striking features, its significancy will as little escape observation as its existence.

Facial Anæsthesia in some instances comes on gradually; in others its occurrence is sudden. Neuralgic pain, or a condition of local hyperæsthesia, may precede its development; while facial palsy and facial Anæsthesia are occasionally associated.

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[1] Muskelanästhesie.

[2] Anästhesie des Quintus, Lehrbuch der Nervenkrankheiten.

(*e*) and (*f*) *Anæsthesia of Mucous Surface, and of the Viscera.*—The morbid condition in such circumstances must depend on a failure of the sympathetic to conduct the impression to the brain; but, as a general rule, impressions made on the ganglionic nervous system are not thus conveyed, and it requires a powerful irritation, or condition of notable hyperæsthesia, in order that a consciousness of their existence should be established. The inquiry into the operation of the organic nervous system is one of very great difficulty, and Romberg has truly remarked in regard to it, "Von vorn herein bekennen wir unsere unbekanntschaft mit diesen Zuständen, die bisher nicht einmal zur Sprache gekommen sind, und deren Forschung mit grossen Schwierigkeiten verbunden ist."

§ II. Diseases of the Digestive System.

A. Diseases of the Stomach.

Disorders of Function.
Atonic Dyspepsia.
Neuroses.
Acute Gastric Catarrh.
Chronic Catarrh.
Chronic Ulcer of the Stomach and Duodenum.
Cancer of the Stomach.
Hæmorrhage.
Hypertrophy of the Walls.
Stricture and Obstruction of the Cardiac Orifice.
Obstruction of the Pylorus, with Dilatation.
Softening.
Perforation.
Rupture.
Tubercle.

DISEASES OF THE STOMACH.

BY WILSON FOX, M.D. LOND., F.R.C.P.

I.—DISORDERS OF FUNCTION.

THE disturbances in its physiological functions which characterise disorders of the stomach present but few characteristic features by means of which those arising from other than organic diseases can be distinguished from those depending on anatomical alterations in its coats. The significance of the symptoms met with must therefore, in the majority of cases, be estimated from the concomitant circumstances by which they are attended, and their treatment must vary with the causes on which they depend. It is not, however, unimportant that these should be briefly reviewed, and in the present chapter some consideration will be given to the causes of such derangements. Their diagnosis and treatment will, however, be more appropriately considered in the subsequent sections which relate to the disorders in which they originate.

A. SENSATION.—*Pain* in the stomach has received various names, the most common of which are Cardialgia, Gastrodynia, or Gastralgia.[1]

The subjective sensations embraced under these terms present all possible variations of degree, severity, and duration, from a vague sense of uneasiness to intense and almost unendurable suffering.

The *seat* of pain, originating in the stomach, presents some remarkable differences. Ordinarily it is epigastric or post-sternal; but it may be felt in the hypochondria or in the umbilicus. Dyspeptic disturbances are frequently associated with pain or tenderness in the dorsal and thoracic muscles, especially about the shoulder-blades. Cancer and ulcer are also very commonly associated with severe dorsal pain, and in some cases more distant neuralgias[1] are attributable to these causes.

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[1] These terms have been applied with such varying meanings by different writers, that it is scarcely correct to speak of them as strictly synonymous, except as being expressions of the common element of perverted sensation. The first two are in most common use in this country, where Cardialgia is more commonly identified with *Acidity* or *Heartburn* (Copland, Dict. ii. 329; Cullen, loc. cit. ii. 465), and Gastrodynia with *Pain*, in the strictest sense of the word. On the other hand, in France and Germany (Georget, Dict. de Méd. art. "Gastralgia;" Romberg, Dis. Nerv. Syst. i. pp. 104, 129; Bamberger, Krank. der Chylopoiet. Syst. 163) Cardialgia (which is the classical expression of the older writers, Hoffmann, J. Frank, Schmidtmann, and Trnka) is used for the severer forms of pain, associated with intense depression and faintness, and is sometimes further limited to those of paroxysmal and spasmodic character; while Gastrodynia is employed for pain of less severity, but more continuous in character. The term Gastralgia, very little employed in this country, is used in France to signify a much wider range of phenomena, but all embracing various forms of uneasiness observed during the digestive process. Barras (Traité sur les Gastralgies et les Enteralgies) defines it in terms corresponding to the "morbid sensibility of the stomach" of Johnson; but, under the theory of the neurotic origin of Dyspepsia, he applies it to almost all forms of indigestion not having an inflammatory or an organic cause. See also Valleix, Guide du Médecin Pract. iv. 3.

There can be little doubt that the mucous membrane of the stomach, though ordinarily possessing but little sensibility, may have this increased under the influence of disease, though the variations in its manifestation under such circumstances are not always explicable. There appear also to be at least two distinct methods through which painful sensation can be produced in this organ; and when it can be made practically available, this distinction is of some importance as a clue to treatment. Thus in one set of cases it may be referred to the direct agency of the sensory nerves of the mucous and submucous tissues; in another, cramp or spasm of the muscular coat may be the determining cause of pain of considerable intensity; while, in a third class, the co-existence of both becomes a matter of great probability.

The conditions of the stomach giving rise to pain may be summarily expressed as follows:—

1. The presence in its interior of foreign substances of an irritating character.
2. Organic diseases altering the anatomical structure of its coats.
3. Perversions of its secretions.
4. Perversions of innervation:
    - *a.* Proper to stomach.
    - *b.* Reflected from other organs.
    - *c.* Originating in the nervous centres.

1. Foreign bodies usually appear to cause pain through exciting spasm of the muscular coats;[2] and this explanation is probably applicable to the pain arising from indigestible food being swallowed, or from bile regurgitating from the duodenum, or from flatulent distension by gas, or from acrid substances arising from fermentation. Sharp substances, on the other hand, may directly injure the nerves of the organ;[3] and the pain arising from corrosive poisons is probably in great measure due to this latter cause.

2. Inflammation, unless when of great intensity, or when caused by corrosive poisons, or when accompanied by aphthous ulcerations or hæmorrhagic erosions, seldom causes pain of extreme degrees of intensity. Very considerable epigastric uneasiness and distress often,

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[1] As of the fifth nerve; Andral, Clin. Méd. ii. 158.

[2] Beaumont, loc. cit. 105, 228, 229, found that cramp-like pain was excited by passing the thermometer into the pylorus in Alexis S. Martin's stomach; but in other cases of Gastric Fistula the sensations produced by touching other portions of the stomach, appear to have been rather those of sickness or faintness than of actual pain. Murchison, Med.-Chir. Trans. xli. 16.

[3] See a case quoted from Velpeau by Dr. Budd, of a fork swallowed, Dis. of Stomach, p. 276. Also a case by Mr. Marshall, where a number of pins were found in the stomach, Med.-Chir. Trans. xxxv. Also a case by Marcet (Med.-Chir. Trans. xii.), of a man who, on several different occasions, swallowed a number of clasp-knives.

however, attend this process; so that this distinction is only of comparative value.

Cancer and chronic ulcer are amongst the most frequent sources of severe and continuous pain in the stomach. Their prominence in this respect depends in great measure on the invasion of large branches of nerves in the destructive processes which they occasion, or in cicatrical contractions resulting from these, and also on spasmodic action of the muscular coats, resulting from obstructions of the pylorus, and further on the irritating secretions to which they may give rise. They may, however, in exceptional cases proceed to a fatal termination without any appearance of this symptom.

Diseases of the pyloric orifice are causes of pain through the spasmodic contractions which they occasion, as well as by the flatulent distension resulting from the fermentation of the food thus delayed in the stomach.

3. Perverted secretions, when acid, may give rise to pain from their irritating qualities. In some instances, however, the symptom attends the secretion of neutral fluids; and in either case it is probably questionable whether both the secretion and the disordered sensation are not common expressions of a more general cause affecting the innervation of the stomach. The fallacy of confounding acidity from hypersecretion with that arising from fermentative processes must also be guarded against; in the latter case the pain is often caused by spasmodic contraction, arising from flatulent distension.

4. Pain of a purely nervous origin, unattended by undue muscular spasm from flatulent distension, or by perverted secretion, is a comparatively rare event in the history of disorders of the stomach.

Such cases will be more fully alluded to hereafter, when it will be seen that they most commonly occur in cases of hysteria and hypochondriasis, or in patients of a rheumatic or gouty diathesis, or are produced by reflex disturbance originating in other and distant parts. (See chapter on the Neuroses of the Stomach.)

It is an uncertain point whether the nerves which are affected in these cases are the branches of the vagus or of the cæliac axis.[1]

In many cases, especially of chlorosis, the neuralgia appears to depend on the general condition of the system, disappearing with improvement in the state of the blood.[2] Direct evidence of pain of this class originating in the central organs of the brain or cord is rarely afforded,[3] though the absence of any demonstrative cause may at times lead to the suspicion of such a mode of causation. Many also of the painful sensations felt in the stomach are directly due to derangements in other organs. Among those which most frequently produce this effect are especially to be mentioned disorders of the uterus and ovaries, and many severe cases of gastrodynia are connected with disturbance of the menstrual function.[1] To these must be added gall-stones, diseases of the duodenum, abdominal aneurism:[2] and pericarditis.[3]

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[1] Romberg, Dis. Nerv. Syst. Bernard found that after division of the pneumogastric nerves the mucous membrane of the stomach became insensible to pinching (Lec. Syst. Nerv. ii. 424).

[2] Niemeyer, Lehrb. Path. Therap. i. 545.

[3] See a case by Bamberger, loc. cit. 168.

DIAGNOSIS.—The difficulty in the distinction of pain of neuralgic or spasmodic origin from that originating in organic diseases is very considerable. It will be more fully dwelt upon hereafter under the diagnosis of those affections. It may be sufficient here to mention that neuralgic pain is most common in the earlier periods of life, and in the female sex (in whom it is usually associated with uterine disturbances, and also with other nervous phenomena), that it affects but little the function of digestion, and that it is often most felt when the stomach is empty. Pain felt after food has a gravity commonly proportioned to the time which has elapsed before it is perceived. If occurring late, it may be due to flatulence; but pain of any severity, occurring early, and continuing long after the ingestion of food, and particularly when it is relieved by emptying the stomach, is always to be regarded with suspicion of its origin in organic disease. Pain of a continuous character and of a fixed site has also graver features than that which is more migratory and intermittent.

The indications obtained by pressure are of some value in elucidating the cause of pain. That arising from ulcer, cancer, and also the uneasiness felt in inflammatory states of the stomach, is generally, but not invariably, aggravated by this proceeding.[4] Pain of nervous origin, and that arising from flatulent spasm, is, on the other hand, usually relieved by pressure firmly exercised.

Some affections which simulate gastric pain deserve a brief notice:—

(*a*) Pain in the abdominal muscles has been shown by Briquet[5] to be frequently mistaken for severe gastrodynia. He states that it is distinguished by a superficial tenderness, by its preferential seat in the left recti and obliqui abdominis, by its affecting not only the fleshy parts, but also their tendinous attachments, and by its being frequently accompanied by dorsal pain, and by tenderness on pressure in the vertebral groove (rachialgia, Briquet). He is of opinion that it may exist independently of any affection of the stomach, though it may occasionally be excited by reflex sympathy,[6] when this organ is diseased.

(*b*) Rheumatic pains of the abdominal muscles are another source of fallacy.[1] They are distinguished both by superficial tenderness and by pain on movement.

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[1] See Neuroses of the Stomach.

[2] Lebert, Handb. der Spec. Path. Therap. (Virchow), vol. v. Abth. ii. p. 58.

[3] Andral, Clin. Méd. ii. 148.

[4] Much in these cases depends on the seat of the disease.

[5] Traité Clinique et Thérapeutique de l'Hystérie, 1859, p. 216 et seq.; termed by him "Myosalgia" and "Epigastralgia" (corresponding to the Myalgia of Dr. Inman, "Spinal Irritation").

[6] This opinion is in some degree corroborated by Bernard's observation, that pricking the solar plexus and semi-lunar ganglion caused involuntary movements of the pectoral and abdominal muscles and of the diaphragm (Lec. Syst. Nerv. i. 368). I have met with unequivocal tenderness in the muscles of the vertebral groove and in the abdominal muscles in cases of gastric ulcer. Traube also has noticed both hyperæsthesia and anæsthesia of the cutaneous surface in cases of ulcer (Deutsche Clinik, 1861, p. 63).

(*c*) Epigastric pain has also been observed in cases of functional or organic disease of the spinal cord. In the former class of cases, when affecting the skin, it is distinguished by the very superficial tenderness, which disappears on deeper pressure,[2] by the discovery of other painful points in the course of the nerves affected, by the absence of all symptoms referable to the stomach, and by the co-existence of an hysterical diathesis: the distinctive characters of pain affecting the muscles have been already referred to. In the latter class the presence of spinal tenderness, as ascertained by cold, heat, pressure, &c., the co-existence of some perversions of the functions of sensation or of motor power in the lower extremities, and even, in the absence of the latter, the symmetrical character of the affection,[3] and the relief by rest, will generally suffice to indicate (in the absence of symptoms referable to the abdominal viscera) the nature of the affection.

(*d*) Pain in the transverse colon frequently simulates that arising from the stomach. It is often associated with an amount of flatulent distension which may add greatly to the difficulties of diagnosis of its seat. There is, however, generally a distinct difference, especially on gentle percussion, between the notes to be elicited from the two organs; that arising from a distended colon being the less prolonged, and having a higher pitch. Pain also from this source is seldom so much felt at the ensiform cartilage as in the right or left hypochondriac regions, and it frequently extends in the direction of the sigmoid flexure. It is also associated with colicky pains and with irregular contractions, which may be seen or felt by the hand, and with borborygmi, distension, and other signs of intestinal flatulence, and with migratory pains in other parts of the abdomen.

B. MOVEMENT.—Of the functional disturbances in the movements of the stomach, *spasm* has been already alluded to as a cause of pain.

Paralysis of the muscular coat from over-distension or from impaired innervation is one of the causes of Dilatation of the Stomach, and will be referred to under that head.

Vomiting, the other derangement coming under this head which most demands attention, may, like pain, originate from causes intrinsic or extrinsic to the stomach, and it may be conveniently classified according to its origin from sources of peripheral irritation either of the stomach or of other viscera, or from disturbed action of the central organs of the nervous system.

As a reflex act it may arise from irritation of the nerves supplying the stomach and fauces, or of other and distant parts which have no direct apparent connexion with these in their sources of nervous supply. The former scarcely need further illustration, since they are familiarly known, and will be repeatedly alluded to hereafter. The latter require to be borne in mind as sources of error in diagnosis regarding the origin of this symptom. Among the disorders of other organs which most commonly produce it are those of the uterus [1] and ovaries, and of the testicle, renal and biliary colic,[2] hepatic abscess,[3] peritonitis, ulcerations,[4] invaginations, hernia, or other obstructions of the intestines, epiploic hernia,[5] and paroxysmal cough.

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[1] Bamberger, Krank. Chylopoiet. Syst. 171.

[2] This is, however, a peculiarity of some forms of pain undoubtedly originating in the stomach, and which cannot, therefore, be relied upon.

[3] Hilton, Lectures on Rest and Pain, pp. 79, 80.

Vomiting from cerebral causes may arise from cerebral anæmia,[6] and also from nearly all the known disorders or mechanical injuries of the brain, independently of the part affected, as well as from some whose nature is less distinct, as in some cases of commencing paralysis after diphtheria.[7] It is a common symptom at the commencement of apoplectiform attacks in elderly people,[8] and it occurs with great frequency in tubercular meningitis.

Certain toxic agents acting through the nervous centres also produce it,[9] and in the same category must be placed the influence of many disordered conditions of the blood, as in uræmia and in the invasion of the acute diseases,[10] and in the cold stage of intermittents.[11]

It is also associated with other and purely functional disorders of the nervous centres, as when arising from shock or fright. It is a frequent accompaniment of the hysterical diathesis, to which more special allusion will be made hereafter. It is easily excited also by affections of the senses, as by severe pain, by objects nauseous or disgusting to the sight, taste, or smell, or even by the idea of these, and by a bright light. The sight of objects in motion also produces the symptom, in some people. Swinging movements of the body are also capable, when continued, of giving rise to vomiting, with various degrees of facility in different persons; and both of the last-named causes probably combine to produce the phenomena of sea-sickness, which is in many very closely imitated by the effects of riding in a carriage and especially by riding backwards.

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[1] The irritability of the stomach in affections of the female genital organs, so constantly illustrated by the state of pregnancy, should, in cases of doubtful origin, never be overlooked by the practitioner. In a case related by Gooch, from Denman's Experiences, vomiting followed each attempt to tighten a ligature round an inverted uterus, and ceased as soon as the ligature was slackened. Dis. Women and Children, New Syd. Soc., p. 137.

[2] Budd, Dis. of Stomach.

[3] Ib. p. 192.

[4] The author has known severe vomiting depend on this cause.

[5] Chomel, Des Dyspepsies, pp. 133, 134.

[6] Marshall Hall, Med.-Chir. Trans. xiii. Kussmaul and Tenner on Convulsions, New Syd. Soc., pp. 28—30.

[7] Sir W. Jenner on Diphtheria, p. 42. In these cases, from the simultaneous affection of the heart, there was reason to believe that the roots of the par vagum were probably affected.

[8] Abercrombie's Second Form of Apoplectiform Attack.

[9] Tobacco, digitalis (Clarus, Arzeneimitellehre, 596; Andral, Path. Interne, i. 147), opium, cyanide of potassium applied externally (Andral, Clin. Méd. v. 270), lobelia, and the vapour of chloroform. Majendie's experiment, by which vomiting was induced in a dog after a bladder had been substituted for the stomach, by means of injection of tartar emetic into the veins, would appear to show that this agent has a similar effect.

[10] Typhoid fever, pneumonia, and scarlatina are among the most common of these.

[11] Henoch, Unterleibs Krankh. ii. 337. Habershon, Obs. Alimentary Canal, 140.

The diagnosis of vomiting arising from disease of the stomach from that depending either on cerebral or on reflex causes is at times very important, lest a grave disease in some other organ be overlooked through the attention being directed solely to the symptoms presented by the stomach. The criteria of the latter class are often by no means distinct, and the origin of the symptom can only be elucidated by a careful investigation of the concomitant circumstances. In some of these cases, however, the vomiting resembles that arising from cerebral causes.

Among the chief differences observable between vomiting arising from the latter cause and that depending on irritation of the stomach, the following deserve attention.

Vomiting arising from disorder of the stomach is usually preceded by nausea, and is attended by more or less epigastric pain and oppression and uneasiness, together with other signs of derangement of the digestive system, such as constipation or diarrhœa, a loaded tongue, and thirst. Moreover, the nausea and vertigo which precede or accompany it are usually relieved by the act of vomiting.

Vomiting from cerebral causes is often unattended by nausea, but vertigo or pain in the head is very common, and both these symptoms are rarely relieved by the vomiting. There is commonly no epigastric pain[1] or uneasiness. The tongue is often clean.

Other cerebral symptoms are also usually superadded, such as pain,[2] diplopia or indistinctness of vision and alteration of the pupils, confusion of ideas, loss of memory, anæsthesia or some form of paræsthesia, spasm, convulsions, paralysis, or coma.

The indications obtainable from the matters vomited vary considerably in their diagnostic value. Blood will be more fully considered hereafter. Food may either be returned unaltered, as in some cases of nervous vomiting, where it is ejected almost as soon as it is swallowed; or it may present evidences of fermentative changes, varying with the length of time during which it has been delayed in the stomach, but which more particularly affect the starchy substances. These are in some cases changed into a tenacious glutinous material, resembling some of the products derived from the lactic acid fermentation,[3] while in others they are found to be frothy from the evolution of carbonic acid, and associated with the formation of large quantities of the *Torula cerevisiæ*.[4] In other instances alcohol[5] has been found, together with amylic alcohol and the butyric, lactic, and acetic acids. These changes occur in the most extreme degree when the food is delayed in the stomach by obstructions at the pyloric orifice, under which circumstances the *Sarcina ventriculi* of Goodsir is found in the scum which rises on the surface. This growth assumes the form of oblong plates, divided by dissepiments into four secondary, sixteen tertiary, and sixty-four elementary square cells, which measure from $\frac{1}{800}$th to $\frac{1}{1000}$th of an inch along each of their sides; and from the arrangement thus described it received the name of *sarcina* or woolpack, given to it by its discoverer.[1]

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[1] Exceptional cases occasionally occur. Some of these have been quoted by the author in a former work, Diagnosis and Treatment of Dyspepsia, p. 60.

[2] Disorder of the stomach is often accompanied with severe cerebral pain, especially under the form of sick-headache, which is sometimes associated with much intolerance of light. It is, however, usually relieved by vomiting.

[3] Frerichs, in Wagner's Handwörterbuch der Physiologie, art. "Verdauung," p. 804.

[4] Ibid.

[5] Graham, quoted by Sir William Jenner, Med. Times and Gaz., Aug. 1851, p. 192; also Schulzen, Arch. Anat. Phys. 1864, pp. 491—498.

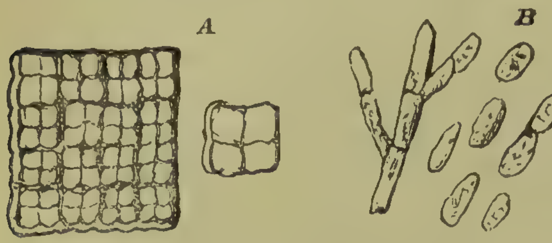

Fig. 1.—*A*, Sarcina ventriculi. *B*, Torula cerevisiæ.

Though, however, for the reasons just stated, its appearance is most common in cases of pyloric obstruction, it is by no means pathognomonic of this condition. It has been found in many other diseases of the stomach, occurring in several cases of direct injury to this viscus,[2] and in some instances where digestion was probably affected by impaired innervation, and when no lesion of the organ has been discoverable.[3] It occurs also in cases of catarrh of the stomach,[4] and probably in other conditions when fermentative action is constant or frequent, though it appears to be doubtful whether it is capable of causing this action.[5] Its generation in the stomach appears to be mainly associated with an acid form of fermentation (Budd); but it has been found in other tissues and fluids of the body,[6] though the conditions of its formation under these circumstances have not been fully explained.

Independently of food, other matters vomited are sometimes of value in the indications which they afford of disease of the stomach. Thus mucus is almost invariably an evidence of catarrhal conditions; while with respect to other fluids, whether acid or alkaline, the chief conditions under which they are secreted are alluded to under the heads of Pain and Acidity.

Bile commonly appears whenever the straining is long and violent;

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[1] Ed. Med. Surg. Journ. vol. lvii. The growth appears to be identical with the *Merismopœdia punctata.* (Mettenius, Zeitsch. Rat. Med. vii. 355.) It is termed *Merismopœdia ventriculi* by Robin.

[2] Mr. Busk, Microscop. Journ. 1841, i. 321; Sir W. Jenner, Med. Times and Gaz. Aug. 1851, p. 192 (Sir W. Jenner's case was probably one of pyloric coarctation resulting from the cicatrization of an ulcer); Budd, Diseases of Stomach.

[3] Busk, loc. cit.; Dr. J. W. Ogle, in a case of Tubercular Meningitis, Path. Soc. Trans. vi. 17.

[4] Dr. Bence Jones, in a case of vomiting from albuminuria, Path. Soc. Trans. iii. 328.

[5] Virchow, Archiv, i. 271. Kühne, Lehrbuch der Phys. Chemie, p. 59, says that he has kept sarcinæ with vomited matters, and also with solutions of sugar, for days, without the slightest development of gases ensuing.

[6] In the lungs, by Virchow, Froriep. N. Notizen, 1846, No. 825; Archiv für Path. Anat. x. 401; also by Zenker, Zeitsch. Rat. Med. N. F. iii. 117. In the urine, by Heller, Archiv Chem. Microscop. 1847, 1852. Also in the pelvis of the kidneys and in the bladder, by Mr. Hepworth, Microscop. Journ. v. 2, 3; and in the bile, by Dr. Lionel Beale (quoted by Dr. Budd, loc. cit.).

it is not therefore indicative of any special disease, though its presence in the stomach serves at all times to retard the digestive process.

Pus, as such, does not appear to be formed in the stomach, except in those rarer instances where suppuration takes place in the submucous coat; and its presence in vomited matters is therefore generally indicative of its having been formed in the œsophagus, or that it has entered the stomach from some source external to that organ.[1]

Vomiting of foreign products, such as worms or echinococci, are among the rarities of medical literature: in the case of the latter their appearance would be indicative of a communication having been established between the cavity of the stomach and an echinococcus cyst in some other organ.

It is believed by some practitioners that cancer cells can be distinguished among the products of vomiting when the disease affects the stomach. I know of no authentic instance where such an observation has been made of an indubitable kind;[2] while the improbability that any portion of a cancer would be seperated in a condition in which its cells would still present their distinctive characters, coupled with the fallacies presented by the appearances of swollen epithelial cells from the stomach, or from the buccal, pharyngeal, or œsophageal surfaces, should cause such evidence to be received only with the greatest care and caution.

Dr. Quain and Mr. Beardsley have, however, recorded a case where a polypoid growth having all the characters of those ordinarily found in the stomach was ejected entire;[3] but the pedunculated growth of these formations would allow of their separation with much greater ease than in the case of cancers, portions of which are ordinarily only removed by sloughing processes, which destroy the characters by which they can be recognised.

Fæcal vomiting can as a rule only occur under conditions either of direct communication of some portion of the intestine (usually the colon) with the stomach, or as the result of obstruction to the passage of the fæces through the intestines. Briquet has, however, recorded a case, observed under circumstances where all possible precautions appear to have been taken to guard against imposition, and where a hysterical patient speedily vomited matters introduced into the rectum.[4]

The contents of the stomach are sometimes ejected by the simple contraction of its coats, without the participation of the abdominal and thoracic muscles; and to this process the name of *eructation* has been applied. Gas is very commonly ejected in this manner, and it is often accompanied by small portions of food. Acid or neutral fluids are also frequently thus brought into the mouth. The vomiting of infants very closely resembles this process, as from the shape of the stomach in infancy its contractions have a greater tendency to force food through the cardia than is the case in the adult, when it is propelled into the fundus. This effect under any circumstances is commonly due to undue irritation or to excessive distension of the organ, and therefore is very common when food is swallowed without due mastication. In some persons the continuance of the habit has led to a habit of *quasi-rumination,* the food thus brought up being subjected to a second mastication before it is swallowed.[1]

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[1] See a case of this nature, where pus was vomited from a fistulous communication between the œsophagus and pericardium, Dr. Chambers, Indigestions, p. 175. The same author says that he has seen this product in matters vomited in cases of cancer of the œsóphagus and cardia.

[2] Lebert, Traité des Maladies Cancereuses, p. 304, says that these have been observed by M. Schoenhein (Qy. Schönlein), but gives no further particulars.

[3] Trans. Path. Soc. viii. 219.

[4] Loc. cit. p. 315.

C. SECRETION.—Alterations in the secretions of the stomach may be classified under the categories of excess, deficiency, or perversion.

It must, however, be remembered that secretions both of an acid and also of an alkaline character are normal products of the mucous membrane: the former being furnished by the glands situated in the fundus and body of the organ, which are lined by a spheroidal epithelium, and which furnish the true gastric juice; the latter by those of the pyloric region, whose epithelium is columnar, and whose products, having the characters of ordinary mucus, possess little or no digestive properties.

It is important, however, that it should be borne in mind that the presence of an excessive amount of free acid in the stomach is not in the majority of cases due to excessive secretion from its coats, but depends with great frequency on unnatural changes in the food; so that it is necessary at all times to distinguish *the acidity which results from fermentation* from *the acidity resulting from secretion.*

The former is due to all causes tending to delay or pervert the normal changes which the food undergoes in the digestive canal, and hence it is a common symptom of indigestion, however produced; it often also appears in its most intense forms when the natural secretions are diminished, or when their amounts do not present their normal relation to one another. Hence acidity from fermentation may arise (1) in all cases when digestion is delayed from imperfect supplies of the gastric juice; or (2) when the food is merely retained in the stomach by obstructions of the pyloric orifice; or (3) when food in a state of fermentation is introduced into the stomach in quantities sufficient to neutralize the antiseptic action of the gastric juice; or (4) when mucus possessing a catalytic action is secreted by the stomach; or (5) when, from disordered conditions of the salivary and buccal secretions, the proper changes are not effected during mastication in the amylaceous portions of the food; or (6) when in otherwise healthy subjects an excess of starchy or amylaceous substances is taken with the food. The results of this fermentative action are shown in the formation of acid products, usually derived from the starchy and saccharine elements. The acids so produced, and which are often formed with great rapidity, are chiefly the lactic, butyric, and acetic, which appear in great abundance under such circumstances: and further evidence of the fermentative character of the process is seen in the evolution of gas, which consists principally of carbonic acid and volatile carbo-hydrogens, while sulphuretted compounds appear when articles containing this element in excess have been taken with the food.[1]

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[1] For further details on this subject see Copland's Dictionary, art. "Rumination."

Flatulence and acidity are therefore almost constant results of imperfect digestion; but though produced by the same causes and therefore probably co-existing in the digestive tract, one only of them may in many cases be made apparent by eructation. Flatulence also may appear chiefly in the intestines, as the result of imperfect changes which the food has undergone in the mouth and stomach. Similar fermentative changes, evidenced by distension of the bowel and by frothy and pultaceous stools, may also result from defective metamorphosis of the food in the lower portions of the intestinal canal, arising from deficient supply or abnormal states of the secretions of the intestines, the liver,[2] and the pancreas, as well as from conditions impeding the absorption of the peptones.

With respect to alterations occurring in the secretions—

1. *An excessive secretion* from the glands of the stomach may affect either of the classes before alluded to, and may furnish fluids either of an acid or an alkaline character. The circumstances under which these different formations occur are not in all cases accurately known to us, but in some cases we are able to distinguish the cause of an arrest of the one and an excess of the other. Thus, under the influence of inflammatory action, or in many cases of excessive physiological stimulation, the acid gastric juice may be either completely arrested or is greatly diminished in amount,[3] while there is an undue formation of mucous secretions of an alkaline[4] reaction.

The most common causes of excessive secretions are, however, those of nervous origin, though the nature of the influences under which they thus originate are only imperfectly understood; and acid and alkaline secretions appear to be produced by different patients under circumstances in other respects apparently similar.

One form of disorder in which alkaline or neutral fluids are furnished in great quantities has received the distinctive name of *pyrosis*, from the burning sensation at the cardia which often precedes its ejection. It occurs endemically among the poorer classes of Scotland and of certain northern countries, and is, under these circumstances, attributed to the effects of an irritating or of an innutritious and insufficient diet. It is met with, however, occasionally, in the wealthier classes of both sexes, but particularly in nervous females, when there is no evidence of organic disease; and it also is an accompaniment of ulcer and cancer of the stomach.

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[1] Frerichs, Rep. Clin. Lect., Med. Times and Gaz. 1841, ii. 410, divides the fermentative processes which may take place in the stomach into the following:—

(*a.*) Alcoholic.
(*b.*) Lactic.
(*c.*) Butyric.

The lactic, which is a simple acid fermentation, causes no evolution of gas. The butyric succeeds the lactic, and is associated only with the formation of carbonic acid; but as butyric acid is volatile, it is perceptible in the eructations. Alcoholic fermentation may be attended by the formation of acetic acid and give rise to acid eructations. The conditions of fermentation are the delay of absorption and the presence of mucus in the stomach.

[2] Dogs in which a biliary fistula has been established pass a large amount of offensive flatus per anum.

[3] Bernard, Arch. Gen. Suppl. 1846, p. 7.

[4] Bidder and Schmidt, Verdauungs-saefte, p. 40; Frerichs, Wagner's Handwörterbuch der Physiologie, art. Verdauung, p. 788; Blondlot, Traité Analytique de Digestion, p. 213; Beaumont's Exp. and Obs., Combes' Edition, p. 182; Corvisart (Longet's Physiologie, 1861, i. 184).

The fluid thus ejected is clear, often insipid and neutral in reaction, and it is frequently brought up in considerable quantities by an act of regurgitation, sometimes without any effort at retching, though it may occasionally be expelled by vomiting. It has been considered by some authorities to consist merely of saliva which has been swallowed, an opinion which has received some support by the observation of Frerichs,[1] that it sometimes contains a considerable amount of sulpho-cyanide of potassium. It appears, however, to the author, that this admixture may be due either to some saliva swallowed or mingled with the fluid in the act of ejection. Patients also who are subject to the affection are by no means conscious of an amount of salivation at all proportionate to the quantity of fluid brought up from the stomach; and it would therefore appear more probable that the larger proportion originates from an altered secretion of the fluids of the stomach.

Though the reaction of this fluid is very commonly neutral, yet occasionally highly acid fluids are ejected, under circumstances similar to the foregoing, and when, from their character and reaction, it is probable that they have been secreted by the stomach.[2] Some observations made by Dr. Pavy on these fluids tend to show that, while the acid fluids possess digestive properties, the neutral fluids have, even after acidulation, very little of this power.[3]

Acid fluids are also ejected under other circumstances of nervous derangement, chiefly of a reflex character, as in the vomiting of pregnancy, or from diseases of the uterus, or in that which takes place in attacks of renal and biliary colic. M. Chomel[4] has described another class, when, after attacks of cholera, vomiting of acid matters continued with great obstinacy, and in some instances proved fatal. The nature of these cases is, however, obscure. The fluids vomited do not appear to have been chemically examined, and it may be considered doubtful whether the acidity observed was not derived from rapid fermentative action in the food, caused by the unhealthy condition of the stomach, rather than from any direct secretions from the mucous membrane.

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[1] Loc. cit. p. 791.

[2] For a case of this nature the reader is referred to the article on Cancer.

[3] Treatise on Digestion, p. 132.

[4] Des Dyspepsies, "Dyspepsie Acide Grave." These cases, according to M. Chomel, had a peculiar acid odour of the breath.

2. *Perversion* of the secretions of the stomach is known to occur in the course of uræmia and diabetes. In the former case either urea[1] or carbonate of ammonia[2] is found in the secretions of the stomach. Bernard noticed that under these circumstances the secretion was still acid, and also that, instead of being evoked solely by the stimulus of food, it became more or less continuous,—a fact which may assist to explain much of the disturbance of the functions of the stomach which occurs in the course of this affection. In diabetes, sugar[3] is sometimes found in the secretions of the stomach; but we are not acquainted with any special effect which this admixture has upon the act of digestion.

It is not improbable that other alterations may occur in the composition of the gastric juice in certain blood diseases; but of the nature of these we are as yet ignorant. The possibility, also, of changes of a similar nature arising in the course of affections of the liver and pancreas is a subject rather of hypothesis than of direct proof.

3. *Arrest* or *diminution* of the secretion of gastric juice takes place under so many and varied conditions, that it is impossible to do more than briefly to summarise them here. They will be more fully treated of in relation to the special diseases of the stomach. Thus sudden moral or physical impressions, especially those of a depressing nature, exhausting mental and physical efforts, and many diseases of the nervous centres, have all this effect in common. Inflammatory processes, as already stated, produce the same results to an extent proportioned to the intensity of the process. Weakening diseases and physical exhaustion, and privation of food or water, similarly diminish the amount of the secretions of the stomach. Digestion also may be arrested, probably from reflex causes, through disordered conditions of the lower part of the intestinal canal,[4]—as from constipation, from the presence of worms in the intestines,[5] or even from enemata of cold water administered while digestion is proceeding; and the due appreciation of the influence of these and similar etiological circumstances is of the utmost importance in arriving at a correct understanding of many of the causes of an imperfect performance of the digestive act.

D. DIGESTION.—Imperfections in the due changes of the nitrogenized elements of the food by the gastric juices in the cavity of the stomach constitute the most important of the disorders in the functions of this organ.

To these defects the terms of indigestion, dyspepsia, or apepsia, have been applied.

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[1] Lehmann, Phys. Chemie.

[2] Frerichs, die Brightsche-Nierenkrank., p. 97; Bernard, Liquides de l'Organisme, ii. 49.

[3] Bernard, Lec. Phys. Exp. i. 295, 300.

[4] Trousseau, Union Médicale, 1857.

[5] Beau, Dyspepsie Vermineuse.

In treating, however, of such aberrations as among the functional disorders of the stomach, several points require to be distinctly understood. In the first place, indigestion may result from derangements in the action of other parts of the gastro-intestinal canal, which contribute to the metamorphosis of the different constituents of alimentary matter. Disturbances in the digestive act may thus be caused by defects either in the quality or amount of the secretions derived from the buccal and salivary glands, and also from those of the pancreas, the liver, and of the intestines, affecting either the starchy, saccharine, or fatty elements of the food, and also of protein substances which have escaped the action of the stomach.

Secondly, similar derangements may depend not only on disorders primarily originating in these organs, but may result directly from the food supplied being either of a nature incapable of being affected by their secretions, or from its being presented to them in a form on which their action is with difficulty exerted.

Thirdly, it is also to be borne in mind, that the direct effects of this imperfect elaboration are such as are more or less common to the whole class of indigestions, and consist of certain fermentative or putrefactive changes in the food, to which allusion has already been made. There are however no certain and practical criteria by means of which under *ordinary* circumstances we can distinguish from the nature of these intrinsic *changes in the food* the special source of the derangement in which they have originated,[1] since the imperfect performance of any one of the functions of the digestion usually disturbs the actions of those next in physiological sequence, and often, though in a less but varying degree, reacts injuriously upon those which precede it in the order in which the food is presented.

Fourthly, when we confine ourselves to indigestion as met with in the stomach, which is perhaps the most important organ in the series, we find that this disturbance may be produced by nearly every disorder to which the stomach is liable; and that the nature of the affections in which it originates cannot, as such, be discovered solely through imperfect changes in the food, but must be mainly determined by various concomitant circumstances, which may or may not be readily discoverable.

Hence when viewed as a whole the indigestion of food can only be regarded as a *symptom* revealed by a series of phenomena, some of which are the primary results of imperfect changes in the alimentary substances, while others are the more or less remote effects of these. The former, although varying within certain limits, according to the nature of the food, are common to all forms of indigestion however originating; but though of great importance as indicating the existence of this state, they are yet only secondary in diagnostic value to those symptoms through which the state of the stomach in which they primarily originate can be distinguished, and on the recognition of which any successful attempt at a pathological classification and consequent diagnosis must depend.[1] The distinction is not indeed always an easy one, since the acids and gases, produced by abnormal chemical processes in the food taken, almost constantly give rise to other secondary disturbances in all the chylopoietic viscera, which in some cases, and especially through the pain which they produce, are liable to be confounded with those originating in morbid states of these organs. Still, however, the separation of these classes is practically so important, that it is desirable as far as possible to maintain it, although it may be somewhat difficult to determine in which category any single symptom is to be placed when regarded apart from the whole group with which it is associated.

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[1] The author alludes to the more ordinary forms: the presence of the excess of fat, or the absence of bile in the fæces, would of course respectively point to deficiency of the pancreatic or hepatic secretions, or of imperfect absorption by the lymphatics.

In such a mode of arrangement the symptoms arising directly from abnormal changes in the food,—such as acidity, flatulence, eructation, distension of the stomach and intestines with gas, borborygmi, and alterations in the fæcal evacuations,—serve as evidences of fermentative processes, the nature, causes, and effects of which have been already discussed. Those, on the other hand, which may be regarded as more direct signs of disturbance of the stomach (though sometimes only resulting from the former) are weight, uneasiness, sinking, craving, emptiness, or pain of different degrees of intensity, appearing either when the stomach is empty, or at variable periods, after the ingestion of food. With them also must be placed affections of the appetite and thirst, either on the side of excess, or deficiency, or perversion; and in the same category must be included symptoms arising in parts which are more remote, but yet forming part of the gastro-intestinal canal, such as the tongue, mouth, salivary glands, and fauces. Indigestion however may only be revealed by symptoms appearing in other organs, the connexion of whose disorders with that of the stomach is of a secondary nature, and which may be enumerated according to the parts in which they occur, viz.:—

Disturbances of the nervous system, indicated by neuralgic pains of the thoracic and abdominal muscles, weakness and weariness, or painful aching in the limbs; by headache, vertigo, perversions of vision, impaired intellectual activity, loss of memory, depression of spirits, anxiety, fear, morosity or irritability of temper; or by the various forms of hypochondriasis, melancholia, or hysteria; or, in some instances, by convulsive attacks.

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[1] Much of the difficulty and uncertainty in the treatment of dyspepsia has arisen from its being regarded as an individual disease, referred by one class of observers to atony (Cullen), by another to inflammatory conditions (Broussais), and by a third to disturbed innervation of the stomach (Barras). The confusion has been further increased by many writers, especially of the French school, introducing sub-varieties according to the prominence of individual symptoms, which, however, can never serve as a sound basis of classification. A further account of these opinions will be found in the Introduction to the author's work, "Diagnosis and Treatment of Dyspepsia." The most complete historical account of the views entertained respecting disorders of the stomach is that by Dr. T. J. Todd, art. "Indigestion," Cyc. Pract. Med. iii.

Alterations in the urinary secretions, consisting sometimes of excess of watery fluids, or of urea; sometimes of diminution of the total amount, associated with lateritious sediments;—or of variations in its reaction, which sometimes shows an excessive acidity, and at other times is alkaline at the moment of emission, and containing in the latter case an undue amount of phosphates;—or in its contents, which may be abnormal, and exhibit albumen, sugar, cystine, or the salts of oxalic acid.

Disturbances in the generative organs, evidenced by perversions of the menstrual function, or by leucorrhœa, in the female;—or by impotence, priapism, or spermatorrhœa, in the male.

Alterations in the skin, manifested sometimes by febrile heat and suppression of perspiration; in other cases by general coldness and chilliness, especially of the extremities; or by perspiration on very slight exertion:—or by alterations in its colour or texture, which may be earthy or sallow in tint, or dry and coarse;—or by various eruptions, among the most frequent of which are erythema, eczema, herpes, acne, impetigo, lichen, or urticaria.

Alterations in the circulation, evidenced by frequent palpitation, occurring either spontaneously or on very slight exertion; by irregular action and intermission in the rhythm of the heart's contractions; and by weakness or excitability of pulse.

Alterations in the respiration, as shown by dyspnœa, occurring spontaneously, with a sense of load at the chest, or on slight exertion; or by cough, usually dry; or by asthmatic paroxysms.

Alterations in the general nutrition, as shown by anæmia; by emaciation, affecting all the tissues, but especially seen in certain parts, as in greyness or loss of the hair, caries of the teeth, retraction of the gums, and incurvation of the nails, which are thin and friable; or by excessive liability to inflammation, from slight causes, of the mucous membranes, and particularly of the conjunctivæ and throat. To which we may add, in persons predisposed to such affections, a liability to gout or rheumatism, or to renal or pulmonary affections, so that by very common consent pulmonary phthisis has been frequently regarded as a consequence of long-continued derangement of the digestion.[1]

These symptoms are very variously grouped, and sometimes occur with great irregularity; and it is important to note that those affecting the stomach may in some cases be far less prominent than others which, though occurring in distant parts, are still valuable evidence of the primary disturbance which exists in the functions of digestion.

And it must further be remarked, that many of them are not referable to the disorders of the stomach alone, but to perversions of the functions of the lower portion of the intestinal canal.

The secondary disturbances in the nervous system belong in an almost equal degree to derangements of the stomach and of the intestines; for as Beaumont has shown that vertigo was a common effect of irritation in the former, so, on the other hand, clinical experience is constantly demonstrating that this symptom is equally produced by flatulence and other derangements of the functions or condition of the latter. Many also of the moral and emotional disorders, and particularly depression of spirits, irritability of temper, and hypochondriasis, though sometimes undoubtedly originating from an independent and primary disorder of the nervous centres, appear often to result directly from the condition of constipation so frequently present; and though the origin of this is threefold, arising from imperfect changes in the food, diminished peristalsis of the muscular coats of the bowels, and from deficiency of secretion from the mucous membrane, the liver, and the pancreas, it is probably in a great measure to the last-named cause, which must materially affect the composition of the blood, that the nervous phenomena of this class are mainly attributable.[1]

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[1] See especially Schmidtmann (Summ. Obs. Med. iii. 203), Wilson, Philip, and Dr. Hughes Bennett. This subject will be more fully dwelt upon hereafter.

It is therefore of importance for the cure of indigestion that the pathological condition in which it originates should be recognised, and also that the *causes* of such disorder should be discovered, and if possible removed. It therefore appears desirable briefly to summarise the chief of these, reserving a fuller description of their effects and mode of action for the chapters devoted to special disorders.

Causes of Indigestion.

As a healthy performance of the functions of the stomach depends on the harmony of the relations between the stomach and the food, our classification of the causes of indigestion may be conveniently divided into (1) those which depend on unsuitability of the food, and (2) those which affect the physiological functions of the organ.

1. *Dyspepsia from unsuitability of food*[2] may depend on defects in the quality or in the amount of the ingesta.

(*a*.) Defects of quality may arise through aberrations in the proper proportions of the nutritive elements; among which may be enumerated excess or deficiency of any of the normal ingredients, saccharine, amylaceous, proteinous, or oleaginous, or of some of the naturally indigestible materials which form a part of all human diet, and also excess or defect of sapid substances,[3] which, on the one hand, favour the secretion of the digestive fluids, or may when in undue amount exert an injurious action on the tissues of the stomach.

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[1] This subject has been fully treated of by Dr. Chambers, Lectures chiefly Clinical.

[2] The author is compelled, for the sake of brevity, to present this portion of his subject in a very condensed form. For a fuller explanation of this branch of the subject he would refer the reader to Paris on Diet, Brinton on Food, Parkes's Practical Hygiene, or to a summary of these subjects lately published by him, Diagnosis and Treatment of Dyspepsia, pp. 71 et seq.

[3] Under this head are included all condiments; which generally are of value in exciting the secretion of the saliva, and hence favouring the digestion of starchy foods. They have less influence on the secretions of the stomach (see Atonic Dyspepsia), but appear to be useful in some cases where digestive power is enfeebled by what may be relatively termed natural causes, as from old age, or from the effects of hot climates. It is probable, however, that agreeable impressions made upon the sense of taste favour the secretions of the gastric juice.

(*b*) The food may be introduced into the stomach in an indigestible form through defects of cookery,[1] or through imperfect mastication and salivation,[2] or from its having undergone fermentation or putrefaction, or other changes which pervert or arrest the normal functions of the alimentary canal.[3] Alcohol in excess, or in too concentrated a form, retards the action of pepsine. Ice also, or large draughts of cold water, and diluents in excess, may diminish the secretion of the gastric juice by lowering the temperature of the stomach, or may impair its efficacy by undue dilution.

In the same category may be placed idiosyncrasies which certain persons exhibit against particular kinds of food[4] ordinarily reputed wholesome.

(*c*) Errors in the amount of food, particularly on the side of excess, are among the most common causes of indigestion.

The secretion of the gastric juice, at least in a healthy state, seems in some yet unexplained manner to be proportioned to the amount of material required for the repair of the waste of the system; and in the majority of cases, food introduced in excess of this, acts as a foreign body, and undergoes fermentative or putrefying chemical changes; or in the comparatively few instances where these do not ensue and the food is digested and assimilated, it gives rise to obesity and other evils, on which it would be beyond our province to dwell.[5]

Irregularity in, and especially too small intervals between, the periods of meals, involving the taking of food before the preceding supply has been digested and removed from the stomach, is another most frequent, and when indulged a necessary, source of indigestion. It is well known that the digestive powers of a given amount of the gastric secretion are limited, though the absorption of the peptones already formed, while the pepsine is retained in the stomach[6] by the dialytic action of the mucous membrane, greatly extends the duration of its action. It is nevertheless evident that, if as much food has been taken at one meal as the stomach can digest, the addition of a fresh supply before the former has passed from its cavity will only delay the changes which the whole has under such circumstances to undergo. Some of the food has been shown by Busch's observations[7] to pass rapidly into the duodenum; but a period of nearly five hours[8] must elapse before the whole of a full meal has passed through the pylorus; and in addition to this it is most important that a period of rest varying from one to two hours should be allowed to the organ. There are few medical men who are not acquainted with patients who allow a much shorter interval than this between each of the three principal meals of the day; and the effects of such a system are as injurious as, and practically are identical with, those of excessive eating; though it is often difficult to convince those who indulge in it of the error in their habits.[1] Many delicate people think that it is necessary to eat often to keep up their strength, but fail to recollect that when meals are taken frequently each should be small, and that when meat is eaten three times daily in tolerable quantities, the addition of milk, eggs, wine, and beef-tea, in the intervals, destroys the beneficial effects of all. It must be borne in mind also, in estimating the effects of a given diet on the health of a patient, that the amount required varies with the expenditure of the system, and that a sedentary life, whether habitual or suddenly entered upon, necessitates a reduction of the food taken, if health is to be maintained. The breach of this rule is often observed, and the ill effects produced on those who, having previously lived an active life, retain their customary habits of eating in periods of enforced or voluntary idleness, has long been well known. Indeed, in all dyspeptic derangements it is important for the practitioner to be on his guard against possible errors in diet, and especially against those which, having become a question of habit, and of relative rather than absolute quantity of the food taken, are the more likely to elude both his own and his patient's observation.

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[1] Especially apparent in the amylaceous substances.

[2] The last named of these causes may arise solely from the former, or from disordered conditions of the salivary and buccal secretions, which may either be defective, or, even when abundant, may be too watery or acid.

[3] Impure water, especially such as contains an excess of calcareous or magnesian salts, may be included in this category. See Parkes's Pract. Hygiene.

[4] Milk, eggs, cooked butter (Chomel, Dyspepsies, p. 8), mutton (Sir T. Watson, Princ. and Pract. of Physic, ii. 457), honey (Andral).

[5] See Dr. Parkes's excellent summary of these conditions in his treatise on Pract. Hygiene.

[6] See Kühne, Lehrbuch der Phys. Chemie, 39.

[7] Virchow's Archiv. xiv.

[8] Weber and Budge, loc. cit.; Kühne, loc. cit.

Deficiency of food, though often mentioned among the causes of dyspepsia, and unfortunately holding among these but too prominent a place in the poorer classes of society, acts probably only in an indirect manner on the digestive powers by weakening those of the whole system. In these cases the food is also too frequently of innutritious quality, and the sloppy tea and bread which forms so large a proportion of the diet of many of our hospital patients is a constant source of the flatulent disturbances from which they suffer, and which, under the circumstances in which they are placed, are often very difficult to relieve. The effects of starvation, and the dangers of indiscriminately giving nourishment in such cases, are familiar to all readers of physiological works, and will be again referred to.

2. *Causes of indigestion referable to pathological states of the stomach* may be referred to abnormalities either in its secretions, or in its movements, or in the absorption of the peptones.

With disorders in the last-named of these functions we are only imperfectly acquainted,[2] but it is probable that under certain circumstances their unfavourable influence is by no means inconsiderable.

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[1] See a case in point by Sir T. Watson, loc. cit. ii. 450.

[2] A case of affection of the lymphatics, in what appears to have been a simple ulcer of the stomach, is recorded by Andral, Clin. Méd. ii. 110.

Causes affecting the secretion of the gastric juice are those which play the most important part in this category. They have been already in part alluded to, and may be briefly summarised in the following scheme:—

I. Causes tending to produce Deficiency.

A. *Organic* .
- (1) Inflammatory conditions, which reduce the amount of peptic fluids, but increase the mucus secreted.
- (2) Congestion.
- (3) Degeneration and atrophy of the secretory glands.
- (4) Pyrexial conditions of the system.

B. *Inorganic.*
- (1) General weakness.
- (2) Disordered blood states.
- (3) Disorders of liver and pancreas (?).
- (4) Agencies operating through the nervous system—moral, intellectual, shock, exhaustion, invasion of acute diseases, narcotic remedies.

II. Causes tending to produce Excessive or Perverted Secretions.

(1) Ulcer and cancer.
(2) Inorganic agencies operating through the nervous system, chiefly sympathetic, and depending on irritation of other organs, as the mouth, fauces, intestinal canal, liver and gall-bladder, kidneys, and uterus.
(3) Disordered blood states.

Derangements in the movements of the stomach may injuriously affect digestion either by their deficiency, irregularity, or excess.

Deficiency of movement may result from weakened muscular power, from dilatation, and from paralytic conditions induced either through impaired innervation, or by inflammation of the peritoneal and possibly of the mucous coats.

Irregularity of contraction may result from obstructions of the pyloric orifice, from thickenings, indurations, malformations, or displacements of the stomach, or adhesions to adjacent viscera, or by simple pressure on the organ when distended with food, such as is exercised in certain trades, or by tight stays, or by diseases of the liver.

Excessive contraction induced by undue nervous excitability or by minor degrees of inflammation tend, independently of vomiting, to expel the food too rapidly from the stomach, and in the same category must be placed the effects of destruction by disease of the pyloric ring.

It will be seen from the foregoing that indigestion is in probably a considerable majority of cases the result of pathological conditions of the stomach which vary widely from one another in their essential nature and in their etiological relations. It is therefore impossible to form logical categories of classification founded on the derangements so produced in the functions of the stomach, since all of these—vomiting, pain, flatulence, acidity, &c.—may each in turn depend on many and totally different causes, some of which are organic and some purely functional in their nature. Nor can any convenient classification according to the causes of indigestion be adopted, since even in those forms which arise from imperfections in the manner in which the food is presented, the result is frequently a complex one, and secondary effects of an inflammatory nature are often thus produced in the mucous membrane of the stomach which surpass in importance of intensity and duration the primary disorder of function. A classification therefore on a pathological, and as far as possible on an anatomical, basis, is the only one which can conduce to certainty of diagnosis, or to scientific principles of treatment.

The main lines of division which will be here adopted will be to distinguish the non-inflammatory dyspepsias from those which depend on inflammatory changes; the disorders produced by ulcer and cancer standing again in a distinct and separate category. In the first-named class will be included dyspepsias from simple weakness, whether general or local, and with these will be classed those forms resulting from atrophy, or from simple degeneration of the secretory glands, to all of which, collectively, the term of atonic dyspepsia will be applied.

Disorders in the functions of the stomach distinctly referable to impaired innervation will form a separate category, and the inflammatory varieties will be considered under the heads of the acute and chronic forms.

II.—ATONIC DYSPEPSIA.

ATONIC DYSPEPSIA, which corresponds to the *Dyspepsie apyrétique* or *asthénique* of Broussais, may be defined as a disorder of the digestion, almost invariably chronic[1] in its course, unattended by fever, and rarely, when uncomplicated, by abdominal pain; whose existence is indicated by weight, uneasiness, and languor following the ingestion of food, together with a general depression of the vital powers; and whose causes are, in a great measure, identical with those which induce a general impairment of the nutrition and the powers of the whole body.

ETIOLOGY.—Among the predisposing causes of atonic dyspepsia may be mentioned *hereditary disposition*, the evidence of which, though not resting on absolute numerical data, is so well attested by numerous observers, and, the author may mention, by many instances within his own knowledge, as to be, he believes, incontestable.

Age exercises an important influence also in determining this disorder, the diminution of the power of the stomach at advanced periods of life being, in many instances, as apparent as is that of the muscular or nervous system—a fact which it is of great importance to recollect in the hygienic treatment of elderly people. Nor should the relatively limited digestive powers of the opposite extreme, in early infancy, be forgotten when, in spite of its great assimilative power, the capacity of the stomach for acting on other than a milk diet is extremely small. Aberrations in diet at this period are, however, more frequently the causes of acuter diseases of the gastro-intestinal canal than of simple failure in the metamorphosis of the food.

Impairment of the functions of the stomach may also be more directly produced by *states of the system* associated with more or less permanent conditions of *depressed vitality*, such as are sometimes observed from the effects of hot seasons and relaxing climates, unattended by any appreciable condition of disease. Still more commonly they are found to result from other causes acting either through the nutritive fluids, or the nervous system, singly or conjointly. In some cases alterations in the blood may be apparent, as in anæmia and chlorosis; but in others, even when nutrition evidently suffers, these are less evident. Similar effects on the digestion may be due to exhausting discharges, hæmorrhages, leucorrhœa, profuse suppuration, cancer in other organs than the stomach, indulgence in venereal excesses, loss of sleep, sedentary occupations, especially such as are associated with deficiency of light and air, long-continued and depressing moral emotions, and the *ennui* of insufficient occupation, mental or physical.[1]

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[1] Some writers, and especially the late Dr. J. Todd, Cyc. Pract. Med. iii. art. Indigestion, describe an acute form of atonic indigestion, in which there is a sudden and total arrest of the digestion, attended with symptoms of irritation of the stomach. It may, indeed, be induced by sudden impressions made on the nervous system during the act of digestion; but unless under these circumstances the contents of the stomach are at once evacuated by vomiting, they prove a further source of irritation, and give rise to a condition of more or less inflammatory action, corresponding closely in character to the "Embarras Gastrique" of the French, and which will be more properly treated of among the "Inflammatory States of the Stomach."

Simple loss of functional power is also produced by causes immediately affecting the stomach, such as excess of fluids taken at meals, especially when drunk warm, or by the abuse of narcotics, and of tea and coffee, by prolonged fasting,[2] by the undue use of condiments, which diminish the readiness with which the stomach is affected by its ordinary stimuli, by habitual constipation, and by undue mental or bodily exertion after meals.[3]

To these causes must also be added the indigestion which occurs in febrile states of the system, in which, as observed by Beaumont on Alexis St. Martin, "food taken in this condition of the stomach (see Pathology) remains undigested for twenty-four or forty-eight hours or more."[4]

It must however be remembered that the digestive power of the stomach apparently varies considerably in different individuals, and that a "weak digestion" does not necessarily mean a diseased state as long as persons with this peculiarity recognise the physiological laws of their nature. Health may be maintained by such individuals by regulating their food according to their digestive capacities. Such persons are however seldom able to bear with impunity efforts of mind or body equal to those which are sustained with impunity by individuals of larger powers, inasmuch their digestive power is commonly incapable of compensating for the increased waste. Exceptions however, depending partly on variations of intellectual capacity or muscular or nervous power, are frequently noticed contradicting this proposition.

THE SYMPTOMS of this form of disorder are:—

(*a*) In the alimentary canal. A sense of weight or uneasiness in the stomach after food, occasioned by the slowness of the digestive process—symptoms which are often protracted for some hours after eating, and are frequently continued up to the next meal. This may afford temporary relief, but is in its turn followed by the same train of discomfort. The weight or uneasiness is in some cases felt behind the sternum, giving rise to a sense of dyspnœa, and at others to a feeling as if some foreign body were present in the œsophagus. It rarely, if ever, amounts to pain, though the condition in some patients, especially in anæmic, chlorotic, or hysterical women, may be accompanied by the complication of intercurrent neuralgia, and in others by an excessive amount of flatulence, giving rise to various degrees of gastrodynia or colic. Tenderness of the abdomen is as a rule entirely absent, and pressure not unfrequently gives relief to such pain as may be present, especially when this arises from flatulent distension.

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[1] One digests with the legs almost as much as with the stomach. (Chomel, loc. cit. p. 58.)

[2] The author has known severe and obstinate dyspepsia of the atonic kind induced by the habit of going without food from an early breakfast to a late dinner, and the cause appears explicable by the observations of Bidder and Schmidt, who found that the secretion of gastric juice was markedly diminished after long fasting. (Die Verdauungs-saefte, p. 41.)

[3] This is not unfrequently a cause of more acute attacks, and will be referred to hereafter.

[4] Experiments and Observations on the Gastric Juice, Combe's edition, p. 92.

The digestive powers are about equally impaired both for protein and amylaceous substances, but in many cases the former, when in any excess, aggravate this condition; and oily and fatty matters are very prone to disagree, as also do soups and broths.

Further disturbances appear in the form of flatulence and of eructation, both of gas and also of portions of undigested food, which are often rancid and offensive (probably from butyric acid fermentation). They usually occur some hours after eating—differing in this respect from the acidity following immediately after the ingestion of food which is observed in some cases of nervous origin. The flatulence affects the stomach and bowels in about equal degrees of frequency, sometimes appearing in one more than in the other; but the fermentative changes originating in the imperfect elaboration of the food in the stomach are usually continued in the lower portion of the canal, the functions of which are generally simultaneously impaired.

Impairment of the appetite, though not invariably observed, is a very common feature of all the more marked forms of this complaint. There is frequently a disrelish for food of all kinds, even when exhaustion is felt for want of it.[1]

Thirst is generally almost entirely absent, at least to any abnormal degree. In many cases there is a positive dislike for fluids, which not unfrequently (especially when taken with meals, or when nutriment is presented in a fluid form) are found to aggravate the dyspeptic symptoms.[2] The saliva is said in some cases to be increased (Todd); but this, as far as the author's observation extends, is not the rule in the state now being described.

The tongue is broad, pale, and flabby; pitted at the edges by the teeth; sometimes thinly furred; but becoming more thickly coated when irritation supervenes. The inner side of the lips and gums are pale, flabby, and sometimes spongy; the tonsils are occasionally enlarged, and the uvula and throat relaxed, giving to the voice a thickness and huskiness which the patient sometimes attempts to relieve by hawking and spitting. The breath is not unfrequently heavy and offensive, but not nearly to so marked a degree as in some of the irritative forms.

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[1] A very marked effect of long fasting is familiar to all under the title of having "overstayed the appetite."

[2] "Dyspepsie des Liquides" of Chomel. This fact is easily explicable when we recollect that undue dilution greatly impairs the efficiency of the gastric juice, and that in the condition under consideration this secretion is naturally defective in power.

Constipation forms another prominent symptom; and though primarily resulting from the participation of the secretions and muscular actions of the intestinal canal in the general condition of atony, it aggravates the state of the stomach in the manner before described, and frequently increases the anorexia. The delay of the fæcal matter in the intestines also favours the liberation of gas, which, distending the weakened muscular coat, tends still further to diminish its propulsive powers; hence various circumscribed swellings frequently appear in the abdomen, giving a mingled tympanitic and dull resonance on percussion, and borborygmi are heard when by pressure or the irregular contraction of the bowels the gas is moved from place to place. These distensions are particularly liable to occur in the course of the colon, especially at its flexures in the right and left hypochondriac regions, where they not unfrequently cause a sense of fulness, tightness, pain, and dragging, which are often, though improperly, referred to the stomach, liver, or spleen. Pain from this cause is also occasionally felt in other parts, especially in the left side, at the insertion of the diaphragm into the ribs, or in the scapular regions.

The evacuations may be solid, dry, and hard, or scybalous, or frothy from fermentative action. They are usually paler than natural, and sometimes offensive; but unless an excess of animal food has been taken, this latter character is not so common as in some forms of inflammatory dyspepsia.

Diarrhœa when irritation supervenes may occasionally alternate with the constipation, but the latter is usually the most prominent symptom.

The circulation is depressed; the pulse is weak, soft, and easily compressible—slow when the patient is in repose, but easily excited on the slightest exertion. Palpitation is common; it occurs irregularly, and independently of organic disease of the heart, and is easily induced by slight exertion, but it frequently arises spontaneously without previous physical or mental effort. It is in many cases directly traceable to flatulent distension, though occasionally it occurs without any distinct evidence of this state. Intermission of the cardiac action, though not so common as palpitation, is also not unfrequent.[1]

Dyspnœa is a result not only of the cardiac state last described, but frequently occurs independently of it. The feeling is one of load or oppression in the upper part of the chest, and especially across the middle portion of the sternum, impelling the patient to sigh or to draw a deep breath, in order to relieve this sensation, which, however, speedily returns. Cough is occasionally caused by an elongated uvula, but the irritative cough frequently described as caused by disorders of the stomach is not usual in this form of dyspepsia.

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[1] It may be noted here that a large number of patients who believe, from the symptoms here described, that they are suffering from disease of the heart, are only subject to this form of dyspeptic derangement.

Except in cases where indigestion is directly due to a febrile state, the course of this affection is singularly free from pyrexial symptoms; the skin is soft, flabby, clammy, and moist, and the extremities are frequently cold, particularly after meals. The complexion is often pallid, sallow, and muddy.

The nutrition suffers more by an aggravation of the anæmia or atony which may be present, than by any direct emaciation—a symptom which occurs more rapidly in the inflammatory and irritative forms.

The urine, as a rule, is but little affected; it is usually clear and copious, and contains but little urea, unless some direct causes of enfeeblement of the constitution, producing excessive waste, are present. Under the latter circumstances the urea may be greatly in excess of the normal amount. If irritation supervenes, other changes may occur in this fluid, which will be again alluded to when these conditions are described.

The nervous system indicates in many ways not only its affection by the general state of the system, but also by the special conditions of indigestion. Languor and inaptitude for exertion, and a sense of weariness in the limbs, which are most evident after meals, are often the earliest, and sometimes during a long period the only symptoms of the disturbed functions of digestion. They sometimes pass into an almost unconquerable drowsiness after food, which, when yielded to, affords a heavy but unrefreshing sleep. There is frequently an impairment of the intellectual faculties,[1] which, though more marked during the period of digestion, also continues at other times, and chiefly affects the memory and attention; the temper also is apathetic and timid.

The duration of this condition is very indefinite, depending as it does in a great measure on the persistence of the causes in which it has its origin. If these be removed, and a healthier state of general nutrition be restored, the stomach may, in some cases, easily regain its tone and functions, but in other instances a marked diminution of the digestive powers may persist long after the original cause of disturbance has been removed.[2]

Sometimes the combination of weakened nutrition and diminished functional power conduces directly to further changes, excited by the irritant action of the undigested food on the mucous membrane, and giving rise to phenomena of a more or less inflammatory character. These not only greatly aggravate the original symptoms, but tend materially to prolong the duration of the disorder; the progress of which is frequently varied by acute exacerbations attributable to this cause, and which, it is important to remark, may also be brought into operation by medicinal stimulants administered without sufficient caution.

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[1] Sir J. Clark lays great stress on this diminution of the intellectual powers (Climate, ed. 1830, p. 257.)

[2] See a remarkable case of this kind recorded by Andral, of dyspepsia originating in the habit of masturbation (Clin. Méd. ii. 193).

PATHOLOGY.—The form of disorder of the digestion now under consideration rarely depends on conditions by which the stomach alone is affected. It is with so much greater frequency associated with general states of the system characterised by the terms ATONY or ASTHENIA, that its pathology, in the majority of instances, only forms a part of that of the constitutional states signified by these names, which, if not in all cases capable of precise logical definition, have nevertheless a tolerably distinct meaning. At present they are generally understood to signify a simple impairment of the functional powers of an organ or tissue unattended by appreciable anatomical alterations. In relation to these, however, it requires also to be stated, that as healthy function presupposes and absolutely requires for its performance a healthy condition of nutrition and innervation, so in most of the cases distinguished by simple diminution of power, but unattended by other marked derangements of function, both the nutrition in a molecular sense and also the chemical constitution of the tissues are more or less impaired or perverted. In many instances, however, the nature of these finer alterations is at present unknown; and if in some cases the defect in the vital action is referred to disturbances in innervation, we are only carried back a step further to an inquiry of the same character, concerning the condition of the nervous structures, and where the same as yet unsolved problem confronts us.

MORBID ANATOMY.—Though in a certain number of cases of atony or asthenia the inference of mal-nutrition underlying this state is rather a matter of induction than of positive proof, there is yet a large class where it appears to depend directly on appreciable anatomical alterations of the nature of atrophy or degeneration, the vital phenomena exhibited by which are frequently clinically undistinguishable from those of the former order to which we have alluded.

In this class may therefore be first mentioned those forms of simple atrophy of the mucous membrane of the stomach, associated with marked thinness and transparency of its walls, which have been mentioned by earlier, and even by some modern, writers, with very little reference to their clinical significance, but which, as has been shown by the researches of Dr. H. Jones,[1] Dr. Habershon,[2] Rokitansky,[3] Dr. Fenwick,[4] and the author,[5] are often combined with fatty degeneration and wasting of its tubular structures. These have been found in many instances to have been replaced by a greater or less amount of fibro-nucleated tissue, and this change has been observed by Drs.

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[1] Path. Soc. Trans. iv. and v., 1853-4. Med.-Chir. Trans., 1864. Diseases of Stomach, 1855.

[2] Guy's Hosp. Rep. 3d Ser. ii., 1855. Observations on Alimentary Canal, 1857.

[3] Pathologische Anatomie.

[4] Morbid Changes in Stomach and Intestinal Villi in Persons dying of Cancer: Med.-Chir.-Trans., 1865.

[5] Contributions to the Pathology of the Glandular Structures of the Stomach: Med.-Chir.-Trans., 1858. Diagnosis and Treatment of Varieties of Dyspepsia, 1867.

Handfield Jones, Habershon, and the author,[1] to be in many cases associated with distinct loss of power in the digestive functions during life; while Dr. Fenwick has proved that in such cases the digestive powers of the mucous membrane after death are also markedly diminished.[2]

Both Drs. H. Jones and Fenwick have shown that these conditions may occur independently of inflammatory action. The author's researches have, however, convinced him that such degenerations are produced with great frequency in the stomach, as in other organs, through the destructive effects of this process upon its tissues, and that therefore in many instances simple atony may be regarded as the result of a pre-existent inflammation, though not necessarily indicating its continuance as a cause of the state—a proposition which affords pathological evidence and support to clinical observations on diseases of the stomach, where irritative action has often been known to be followed by long-continued asthenia.

In several of Dr. H. Jones's[3] cases the changes in question were met with at advanced periods of life, and probably belonged to the category of senile degenerations. Dr. Fenwick has also pointed out that in the cases in which he observed these alterations in the stomach analogous degenerations had occurred in other tissues, and especially in those of the vascular system.[4]

Closely allied to these conditions of degeneration are those where the digestive power is weakened and sometimes almost completely abolished by inflammatory or febrile conditions of the system, and of which Beaumont's observations on Alexis St. Martin gave very distinct evidence.[5] Histological investigation also reveals in the stomach changes similar to those discoverable under these circumstances in other glandular organs, as well as in the heart and voluntary muscles. The epithelial cells are granular and disintegrate easily, evincing a tendency to a retrograde metamorphosis, or defective nutrition by which their functions are impaired or diminished. The condition is, indeed, one which it is not always easy to distinguish from the earlier or minor degrees of recent inflammatory change; but there can be very little doubt that molecular softening and granular disintegration may occur independently of this process. Tissues thus weakened in their nutrition are, however, liable to undergo, from the slightest causes, acuter destructive processes associated with vascular disturbance, to which the author believes that the name of "Inflammation" may still, in the present state of our knowledge, be applied; and therefore, while some of the forms of the so-called dyspepsia febrilis belong to simple atony from arrested nutrition, another and a very large class, to which allusion will be made hereafter, require to be classed under those having an inflammatory origin.[1]

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[1] In a former paper of purely pathological investigation into the subject, before quoted, I stated that I had not then had the opportunity of tracing the clinical histories of the patients whose stomachs I had examined. Since that period I have, however, been able to trace in several instances the concurrence of anorexia and loss of digestive power with these conditions of degeneration. Dr. Fenwick has recently published a case—Lancet, 1870, ii. 78—where atrophy of the glands was general throughout the whole of the mucous membrane of the stomach; and where the patient, a gentleman ætat. 45, died with gradually increasing exhaustion. Vomiting also occurred. Dr. Fenwick found the digestive power of the mucous membrane markedly diminished.

[2] In Dr. Fenwick's cases the stomachs so altered showed scarcely any traces of self-digestion, and the mucous membrane, after the addition of hydrochloric acid, when digested with 10 grains of albumen, dissolved only $\frac{6}{10}$ of a grain, whereas 11 grains of albumen digested similarly with a healthy stomach lost four grains.

[3] See especially pp. 96, 107, 113, 126.

[4] Dr. Fenwick's observations were chiefly made upon patients dying of cancerous disease of the breast. As catarrhal conditions of the stomach are very common in cancerous disease, even when this organ is not affected by the growth (see Lebert, Traité des Malad. Canc. 115), the degenerations found by this author may possibly have been the result of past inflammatory conditions.

[5] See also Bernard's confirmation of this evidence, Arch. Gén., Suppl., 1846, pp. and 204.

DIAGNOSIS.—The difficulty in the diagnosis of all varieties of dyspepsia depends on the fact before alluded to, that the symptoms arising from imperfect changes in the food are common to all conditions in which these may originate. The discrimination of the form now under consideration depends chiefly on the absence of the evidences of organic affection and of severe nervous derangement, coupled with the etiological circumstances of its origin. It is essentially a chronic affection, and therefore is mainly to be distinguished from chronic catarrhal inflammation. The leading characteristics of atonic dyspepsia in contradistinction to those of chronic catarrh are—(1) The minor degree of gastric uneasiness, and the absence of epigastric tenderness; (2) the simple deficiency of appetite, and the absence of thirst; (3) the absence of pyrexia; and (4) the condition of the tongue, which exhibits no signs of irritative action of the gastro-intestinal tract[2] (see Chronic Catarrh, p. 894), but is pale, broad, and flabby. The constitutional symptoms are also less severe, and the course of the affection is more uniform.

The diagnosis from cases of ulcer and cancer is easily made when in these affections pain, vomiting, and hæmatemesis are present. In the earlier stages of cancer the symptoms may closely simulate those of simple atony, in loss of appetite and of digestive power, and sometimes it is impossible to obtain absolute certainty in diagnosis. The conditions which would attract suspicion of the more serious disease are failure of appetite, loss of strength, anæmia or other signs of cachexia commencing without manifest cause in a person who has passed the age of forty. Pain, when not traceable to intestinal colic, should always excite suspicion of a more serious disorder than simple functional dyspepsia.

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[1] Beaumont's description is so graphic, and possesses the so infinitely great advantage of being drawn from life, instead of from more dubious post-mortem changes, that it appears well that it should be again quoted *in extenso*, especially as it affords convincing proof that the two distinct conditions of the stomach may exist under these circumstances; the one irritative, the other atonic :—

"In febrile diathesis, or predisposition from whatever cause, ... the villous coat becomes sometimes red and dry, at other times pale and moist, and loses its smooth and healthy appearance; the secretions become vitiated, greatly diminished, or entirely suppressed; the mucous coat scarcely perceptible, the follicles flat and flaccid, with secretions insufficient to protect the vascular and nervous papillæ from irritation."—Loc. cit. p. 98.

[2] For a further discussion of the value of these indications the author would again refer to a previous work, "Diagnosis and Treatment of Dyspepsia," p. 16 et seq.

THE PROGNOSIS of atonic dyspepsia varies with its etiology. Cases where the disorder is of purely functional origin, and where it has been induced by causes acting directly on the stomach, are usually amenable to treatment. When, however, the defective digestion is only a part of a more general condition of the system, the restoration of the powers of the stomach must depend on the possibility of improving the health of the patient, and of removing the conditions which have induced the disease.

Cases also where there is any suspicion of degenerative changes in the glands, as in obstinate atonic senile dyspepsia, or where symptoms of indigestion persist after long-continued inflammatory or catarrhal conditions of the mucous membrane, have necessarily a much more unfavourable aspect than those where the state is one of mere functional inactivity.

When connected with or arising from anæmia, the result of organic disease in other organs, atonic dyspepsia, though seldom immediately fatal, not unfrequently proves a most serious complication of the original disorder, tending, with a rapidity proportioned to its severity, to further impair the strength and the general nutrition of the patient. When uncomplicated it rarely appears to act directly as a cause of secondary diseases, but it seldom continues long without passing into some of the irritative forms, and the effect of these is not only to maintain, but also to aggravate, the original condition of defective digestive power. The injury which it causes to the general nutrition may however predispose to disease in other organs, as the lungs, brain, heart, or kidneys, according to the several liabilities of each of these as a *locus minoris resistentiæ* to other injurious influences which may come into operation; but I confess that I am indisposed to entertain the opinion that simple functional impairment of the powers of the stomach has any tendency directly to induce the more serious organic diseases of this viscus, such as ulcer and cancer.[1]

THE TREATMENT of this form of dyspepsia is comprehended under the title of Tonic, and implies the use of all such agencies as are capable of increasing functional power by restoring the conditions of healthy nutrition; indications which are to be fulfilled by measures adapted—

1. To improve the quality of the blood, and to regulate its transmission, by means of suitable diet, by medicinal agents capable of altering its composition, when this is defective, and by re-establishing the digestive and nervous powers.

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[1] This opinion has been expressed by M. Beau.

2. To enable the system to appropriate and act upon the nutriment conveyed to the tissues, through methods calculated to favour healthy metamorphosis and the elimination of effete products. Our object is thus simultaneously to increase the vigour of the system and also of the stomach; since it must be remembered that, while the due performance of the functions of the latter is essential to the nutrition of the body, yet that, on the other hand, a permanent increase in the powers of any single part cannot take place without a healthy condition of the whole system.

The treatment directed to these objects may be conveniently divided into regiminal and dietetic, and medicinal.

(*a*) Regiminal and dietetic.

As regards diet, the primary necessity is to administer the most easily assimilable food, and at the same time to avoid overloading the stomach, and thus aggravating the existing weakness, or exciting inflammatory irritation.

In cases of great impairment of the digestive powers arising from pyrexial conditions, hæmorrhages, long privation, or exhausting discharges, food must be given in very small quantities at short intervals. Milk, and strong beef-tea, and animal jellies, combined with alcoholic stimulants, are the forms that should be selected for this purpose.[1]

For the less severe but more frequent forms that are met with in the dyspepsias of the sedentary, or of those subjected to mental strain or anxiety, the main outlines of diet only can be sketched in this place. Three moderate meals daily are usually sufficient, but a fourth may occasionally be permitted on retiring to rest. A cup of tea may, however, often be taken with advantage before rising in the morning. The food chosen should be varied, but selected for its digestibility.

Fresh-cooked meat should be eaten at least twice daily. Beef and mutton, and game, with the exception of hares and rabbits, are to be preferred. Chicken, calves' feet, sweetbread, and tripe may also be permitted; but pork and veal, and salted or preserved meats, should be excluded. Eggs, when they agree, are to be recommended.

Fish is less desirable, but may be eaten with moderation. Oysters often are found to agree well; but differences in this respect are observed in individual cases.

Vegetables should by no means be excluded; but caution is requisite in their use. When they cause much flatulence, their place may be supplied by rice or maccaroni, and by some fruits, especially by grapes, strawberries, and stewed prunes. Potatoes should always be well boiled, and not eaten too young. Other vegetables should also be fresh and carefully boiled. Turnips, parsnips, Jerusalem artichokes, onions, and the Cruciferæ often disagree; but spinach, vegetable marrow, beetroot, young peas, and French beans may commonly be taken.

Bread should not be eaten new. Aërated bread often agrees better than the ordinary forms. Biscuits or toast are often, however, preferable. Fresh butter may also be eaten in moderation.

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[1] The methods recently proposed by Dr. Marcet ("On a New Method of preparing Meat for Weak Stomachs") and by Dr. Pavy (loc. cit.), of subjecting animal food to artificial digestion before its administration, may prove to be beneficial in these cases.

Pastry is to be eschewed; but light farinaceous puddings generally agree well. Fried dishes should be forbidden, and in the same category must be placed the Crustacea, and nuts, pickles, and cheese.

Much fluid at meals should be avoided. Cocoa made from the nibs, or milk and water, may be taken when tea and coffee disagree. A moderate quantity of wine should however be taken twice daily.[1] The selection may commonly be left to the patient, unless under special circumstances. Sherry, claret, hock, and champagne generally suit the best. Malt liquors are only to be used with caution, as they often cause flatulence.

Sugar[2] may be used in moderation; but other condiments are to be avoided, except in the case of elderly people, or of those habituated to their use, since they have been shown to possess very little power of increasing the amount of gastric juice, and are liable to cause irritation.

The *general regimen* must also be tonic, including under this head sufficient rest[3] both at night and also after meals; the avoidance of hot, ill-ventilated apartments, both in the day and at night; and the spending as much time as possible, consistently with the avoidance of undue fatigue, in the open air. Travelling, especially in open carriages, yachting, or sea-voyages, frequently prove highly beneficial in cases marked by much weakness, while for those of less severity horse exercise is as a rule more advantageous than walking. Exercise, especially in the open air, pushed to a degree short of producing exhaustion, has probably a greater influence in increasing the digestive powers of the stomach than any other single measure: hence for those who of necessity lead sedentary lives in large cities the use of gymnasia often proves of great service, by bringing into play a larger proportion of the muscular system than is exerted in mere walking. It must, however, be remembered that exhaustion is to be most carefully avoided, and that after any active exercise a sufficient amount of time should be allowed to elapse to allow the body to cool and the nervous system to repose before food is taken.[4]

The influence of the intellectual and moral functions on the digestive powers is so marked that it cannot escape notice in this place; and it should always be recommended to patients harassed by care or anxiety, as well as to those engaged in absorbing intellectual pursuits, to take their meals in cheerful society. In many such cases, however, a complete cure is unattainable, except by a change of thought and scene.

The effects of cold bathing will, as a rule, be beneficial or not according to the powers of reaction of the patient. It is decidedly injurious when this is not speedy, and complete and lasting, and even in some of the latter class the exhaustion following the bath more than compensates for the temporary pleasure derived from its use. A healthy state of the skin being, however, a great object, a tepid bath taken daily, together with the use of the hair-glove or flesh-brush, should be strongly recommended. A residence even temporarily in a dry and bracing climate is frequently of the highest value. The air of Brighton often proves of great service when there is no irritability of the mucous membrane; and next in order may be placed Scarborough, Dover, Folkestone, Margate, Eastbourne, Malvern, Tunbridge Wells, and the Surrey Downs; Ilfracombe, on the western coast, also possesses many advantages. A high situation, on a porous soil, is that which in general is best suited to patients of this class.

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[1] Bernard, Lec. Phys. Exp. ii. 420, has shown that diluted alcohol is one of the most effective stimulants to the secretion of the gastric juice.

[2] Independently of its value as an article of diet, sugar has been shown to be an effective stimulant of the secretion of the gastric juice (Blondlot, p. 223).

[3] The importance of perfect rest is shown by a case of Andral's (Clin. Méd. ii. 191), when a condition of atony supervening upon irritative dyspepsia was only finally cured by retaining the patient for more than two months in bed.

[4] If exhaustion is felt after exercise, it is often advantageous to take a small quantity of an alcoholic stimulant a short time before the food.

As a prelude to all discussion of the effects of medicinal treatment for this disorder, it is not unimportant to state that, although often proving of great value as adjuvants of the general hygienic measures which have now been indicated, the employment of drugs independently of these is of comparatively little service. Even under any circumstances, no little caution is necessary in the selection, doses, and mode of administration of medicinal agents, in order to avoid the causation under their influence of irritative conditions, which are peculiarly liable to supervene in tissues whose vitality and power of resisting external impressions is already below the healthy standard.

The objects to be fulfilled by therapeutic measures may be conveniently summarized under the following heads:—

(1) Tonics to permanently increase the powers of the organ and of the system generally.

(2) Stimulants or stomachics to increase the secretory powers of the stomach, and which, by thus accelerating the digestive process, act indirectly as tonics, by favouring the assimilation of nutritive materials.

(3) Adjuvants to supply materials in which the gastric juice may be presumed to be defective.

(4) Certain remedies for special symptoms or conditions, which may not only hinder the digestive process, but may interfere with the comfort of the patient.

When anæmia is present, the use of iron is strongly indicated; but its action is less marked in cases of functional debility unattended by this state. The carbonate, the potassio-tartrate, the ammonio-citrate, or the ferrum redactum usually suit the best; but when there is much relaxation of the tissues the tincture of the perchloride or the sulphate may be used. The former may be given before meals, the two latter should be taken immediately after food. If these preparations disagree, the lactate or the syrup of the pyrophosphate may be sometimes resorted to with advantage. Parish's acid syrup of iron and lime often agrees well with children, to whom also the vinum ferri is well suited. When constipation is a marked symptom, and also in females whose menstruation is scanty or infrequent, iron may often be combined with advantage with one or two grains of the watery extract of aloes.

In other instances the natural mineral waters of Tunbridge Wells, of Spa in Belgium, or of Homburg, Kissingen, or Schwalbach, prove of greater efficiency than any pharmaceutical preparations; and their beneficial effects are probably much assisted by the advantages resulting from the change of scene and of the mode of living, which those experience who visit these places from distant parts.

It should be mentioned as a caution, that in conditions of gastric irritability iron is usually tolerated with difficulty; and that some preliminary treatment is often necessary before, even in atonic dyspepsia, its administration is attended with advantage. It may often be advantageously combined with mild aperients, of which the aloetic class are generally the best, and particularly in the case of chlorotic females.

Much caution is requisite in resorting to the vegetable bitters, many of which, though increasing the appetite, appear to have little or no influence in augmenting the digestive powers,[1] and, further, have frequently a considerable tendency to cause irritation of the stomach. Their effect as tonics, in any general sense of the term, appears also to be of a very questionable nature.

Two remedies of this class have, however, a more distinct general, and a more persistent local effect, and therefore appear to deserve a place among the true tonics, viz. nux vomica, or its alkaloid strychnia, and quinine. The former of these often proves a most valuable tonic remedy, improving apparently the nervous energy of the stomach, as well as that of the system at large. Thus in many cases, by increasing the muscular contractility of the stomach and intestines, it aids (in addition to the antiseptic effects common to all bitters, but largely possessed by strychnia) in preventing the distension by flatus, which is so common and distressing a symptom in the cases now under consideration.

The most convenient mode of administering it is in the form of the tincture, in doses of from five to ten drops with infusion of orange-peel and syrup; and it may be advantageously combined with the mineral acids, in cases which appear to require the administration of these remedies. Its use in many of the painful neuroses will be further alluded to when they are treated of.

Quinine is a more doubtful remedy in stomach affections, though at times it undoubtedly does good service. Its tendency to cause headache, and nausea and irritation of the stomach, requires caution where any signs of the latter condition exist. Still, in cases of convalescence from severe diseases, when the tongue is clean, but pale, broad, and flabby, and there is little tendency to constipation or to congestive headache, and also in persons of lymphatic constitution with relaxed conditions of the system, it often proves of value. It may be given in pill or powder, in doses of one or two grains, taken daily before or with meals, or in the form of the Tinct. Cinchonæ Composita, or Battley's Liquor Cinchonæ, both of which preparations often appear to agree better than the pure alkaloid. In other cases, where iron is simultaneously indicated, the Ferri et Quinæ Citras may be resorted to.[1]

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[1] See on this subject Buchheim's "Arzeneimittellehre," p. 42; Shrenk, "De Vi et Effectû quorundam Medicaminum in Digestionem;" Diss. Inaug., Dorpat. 1849; Clarus, "Arzeneimittellehre," p. 1014. These authors have remarked that the increase of the appetite is only due to local irritation—an effect which, as Griesinger, Arch. Phys. Heilk. vi. 399, has shown, results equally from small doses of many of the more irritant metallic poisons.

The other remedies of the class of bitters appear to rank rather among the stimulants and stomachics than with the tonics as above described. They may therefore be conveniently subdivided into four sub-classes, viz. simple bitters, aromatic bitters, aromatics, and stimulants [2] to the gastric secretion of a more general character.

The simple and aromatic bitters have the greatest influence in increasing the appetite, and when this is defective their administration is especially indicated, but with the caution that loss of appetite is not always to be relied on as a diagnostic symptom of atonic dyspepsia, but is common in many inflammatory diseases of the stomach, when the use of these remedies is prejudicial. The effects of many of these are familiar enough to the profession, and also to the public, especially in the use of infusion of quassia before meals; and chiretta appears to have a very similar action.[3]

Judging from the researches of Corvisart, it would appear that the aromatic bitters, in addition to their powers of increasing appetite, have a greater influence in promoting gastric secretion than those last-named; and among the principal remedies of this nature may be enumerated absinthe, hop, chamomile, cascarilla, and calumba.

Of these calumba holds the chief place in point of therapeutic value, as a remedy which can be safely employed when others of the class would be too irritating.[4] It may be used as an infusion or tincture,

[1] Quinine more frequently disagrees when given in solution in conjunction with sulphuric acid: a combination rarely indicated, but which, as it appears to me, is too frequently employed.

[2] It is important in relation to this subject that the varied effects of different agents on the gastric secretions should be constantly remembered. Blondlot (Traité Analytique), Bernard (Arch. Gen. 1846), Corvisart (Longet's Physiologie, 1861, p. 184), have shown that the most active agents of this nature are alkalies (and in this respect the effects of the alkaline saliva must be remembered), cold water, diluted alcohol, ether, sugar, absinthe, chicory, ipecacuanha, nitrate of bismuth, and diluted solutions of common salt. To these Corvisart adds black coffee, the effects of which must appear, however, somewhat doubtful to those who are acquainted with the almost total arrest of digestion which sometimes follows its use. Its effect, however, appears to be of an opposite character in different classes of patients, suiting well the lymphatic temperament, but injurious to persons of nervous excitability. See Trousseau et Pidoux, Traité de Thérap. ii. 533. Stronger stimulants, such as cathartics (Blondlot, loc. cit. p. 213; Beaumont, loc. cit. p. 182), powdered salt (Bardeleben and Frerichs, loc. cit. art. "Verdauung," p. 788), condiments (Beaumont and Buchheim and Engel), and even mechanical irritants, as charcoal (Corvisart), produce a large amount of mucous secretion, either alkaline or feebly acid, but possessing but small digestive powers.

[3] Of gentian it should be observed, that though possessing the advantages of a laxative in addition to those of a bitter, its characters as an irritant are more marked, and that additional caution is therefore requisite in its use.

[4] Calumba appears to possess some direct "sedative" properties; thus it is sometimes useful in the vomiting of pregnancy and in sea-sickness, and even in minor degrees of subacute inflammatory action, and it has been known to check the vomiting caused by tartar emetic (Pereira).

and the former may often be advantageously administered in conjunction with other aromatics, or with acids, alkalies, or ferruginous preparations; but when a more active effect of the same character is required, other remedies of this class may be resorted to with benefit.

As regards the more direct stimulants, their administration is indicated before or with food. The effects of a moderate quantity of wine in aiding a weak digestion have been already alluded to; and bitter beer, combining the aromatic and bitter qualities of the hop, often proves of service in milder cases, where flatulence is not one of the symptoms complained of. Where, however, there is a tendency to fermentation of the food, malt liquors are distinctly contra-indicated.

Another remedy of the same class, ipecacuanha, originally prescribed by Daubenton, has of late been brought into more common use, by the advocacy of Dr. Budd. It should be given in pills, in doses of half a grain to one grain before meals, and may often be combined with advantage with three or four grains of rhubarb.

Other combinations of the same kind occasionally prove useful, such as chamomile[1] together with rhubarb and ginger. Cayenne pepper is sometimes employed in the same manner, but its use is less advisable. The value of alkalies when taken during meals, in the form of Vichy water, liquor potassæ, or the carbonates of potash and soda in combination together with wine or malt liquors, is probably in some degree attributable to their physiological effects on the secretions of the gastric juice. They have also been employed for the same object by my friend Dr. George Harley, in small doses, properly diluted, before meals, with good effects in promoting digestion.[2]

As *adjuvants* to the process of digestion two series of agents deserve especial mention, viz. the mineral acids and pepsine.

The efficacy of the former of these has been long known. Their use is not however limited to cases of simple atony, but they sometimes prove serviceable in many cases of irritative dyspepsia when from inflammatory causes the secretion of gastric juice is defective, and to which fuller reference will hereafter be made.

Of these acids the hydrochloric stands undoubtedly the first in point of utility,—a fact which is easily comprehensible from its being probably the most active agent in the normal process of digestion. It should be taken, in doses of from fifteen to twenty drops of the dilute acid of the British Pharmacopœia, suitably diluted with water, immediately before, or during, or directly after a meal. It may be rendered more palatable by the addition of syrupus aurantii; and may often be very advantageously combined with some of the bitter remedies before mentioned, especially with the tincture of nux vomica, the liquor cinchonæ, or the infusion of calumba. Employed in this manner it will often, in the less severe cases, be found to relieve the sense of weight and distension ensuing after food. It frequently also prevents the acidity and flatulence arising from the fermentation which results from the imperfect action of the gastric juice, though it must be considered problematical whether in these doses it has the intrinsic power of checking this process which Liebig[1] attributes to the stronger mineral acids, and to which Pemberton[2] ascribed their influence on the relief of flatulence.

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[1] Chamomile has the advantage of possessing slightly laxative properties, and is said by Trousseau (Traité de Thérap.) to be very beneficial in the atonic and flatulent forms of gouty dyspepsia.

[2] The effect, as Blondlot and Bernard have shown, of the administration of a small amount of diluted alkali on an empty stomach is to cause a secretion of gastric juice, much greater than is sufficient to neutralize the alkali.

The phosphoric and nitric acids have been recommended for the same purposes by other writers (Todd, Pemberton), but their influence in aiding digestion is much less marked than that of the hydrochloric, while nitric acid not unfrequently produces irritating effects on the stomach, and may give rise to pain or nausea.

Dr. Handfield Jones[3] has for similar purposes found the use of lactic acid, as recommended by Majendie,[4] to be productive of good results: he administers it in doses of fifteen to twenty drops, suitably diluted, at meal times, and, in some cases of irritative dyspepsia, he considers it decidedly preferable to the hydrochloric.[5]

Though the efficacy of pepsine has occasionally been called in question by some recent writers, my own experience would induce me to bear a strong testimony in its favour—not only in the form of dyspepsia now under consideration, but also in some conditions when the digestive process is impaired by irritative states of the mucous membrane. It may often be very advantageously taken simultaneously with hydrochloric acid at meal times.

The complications of atonic dyspepsia which most call for medicinal relief are constipation, flatulence, and acidity.

In remedying constipation, much care is required to avoid irritation, and only the gentlest and least irritating laxatives are desirable. When possible, even these should be dispensed with, and the action of the bowels, when not occurring spontaneously, should be daily solicited by the use of enemata of cold water.

Friction, or the wet compress worn at night, protected by a piece of mackintosh, or the use of the cold douche to the abdomen, are often useful adjuvants in this respect.

When medicines are used, rhubarb and aloes are to be preferred to all others. Either may be given with food, a method which diminishes to some degree the danger of irritation resulting from their use, and they frequently may, with advantage, be combined with small doses, as a quarter of a grain, of the extract of nux vomica.

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[1] Animal Chemistry, p. 386. [2] Diseases of the Abdominal Viscera, p. 122.

[3] Functional Nervous Disorders, p. 420.

[4] Formulaire pour la Préparation et l'Emploi de plusieurs nouveaux Médicaments. 1835.

[5] Hünefeld's experiments under Budge, "De Albuminis Succo Gastrico factitio Solubilitate," showed that hydrochloric acid gave the greatest digestive powers, lactic acid less, and acetic acid the least (Canstatt, 1859, i. 30). Trousseau's clinical experience of the relative value of the mineral acids indicates a decided superiority on the part of hydrochloric (Clin. Méd. ii. 377). Meissner also has found that, in artificial digestion, it is necessary to use ten times more lactic than hydrochloric acid to produce a digestive mixture of the same strength (Henle and Pfeuffer's Zeitsch. 3d Ser. vol. vii. p. 16).

Recourse should however be had as little as possible to purgative remedies, for it may become difficult afterwards to shake off the habit of requiring their aid, and the use of strong aperients tends still further to exhaust the muscular and nervous powers of the intestines. In many cases of atonic dyspepsia, when there is any considerable degree of general debility, there is very little occasion for a *daily* action from the bowels, but care should at the same time be exercised to obviate any undue accumulation of fæces in the intestine.

When acidity and flatulent spasm exist together with the constipation, antacids, and especially the magnesia usta, or magnesiæ carbonas, in combination with tinct. rhei and aromatics, may be employed with advantage for the same objects. In severer cases, especially in the gouty flatulence of elderly people or of females at the climacteric period, assafœtida, either in combination with aloes or in the form of the compound galbanum pill, is frequently of considerable service. If the flatus exists in the stomach, large draughts of warm water may sometimes prove efficacious in promoting its expulsion, by eructation;—while tympanitic distension of the bowels may be relieved by enemata containing turpentine, assafœtida, oleum rutæ, or sometimes by infusion of chamomile.

Other remedies for flatulence may at times be employed, though they only act as very temporary palliatives, such as the more cordial aromatics, the spiritus ammoniæ aromaticus, the aqua anethi, or peppermint water. In some cases the absorbent powers of charcoal are of service, though to possess any efficacy of this kind it is necessary that it should be recently prepared.[1]

It must be remarked that no single plan of medicinal treatment should be too continuously protracted, and that a change of remedies within the limits of those above indicated is often productive of good effects. Many that seem at first beneficial appear after a time to lose their efficacy, and may be advantageously replaced by others of a different class according to the predominance of individual symptoms; and even the ferruginous preparation, on which so much stress has been laid, should not be persisted in without intermission.[2]

The period during which bitter remedies prove efficacious is equally limited. Preparations of nux vomica or calumba are tolerated longer than most of the others, but the prolonged administration of the former may entail nervous accidents and dangers of over-excitability of the spinal cord, which may sometimes persist to an unsafe degree after the discontinuance of the drug; and even when its use is beneficial it should seldom be persisted in longer than a month or six weeks. If the favourable effects of bitter remedies do not become apparent after a few days, it is better to discontinue them; and under all circumstances it is necessary to watch closely for signs of gastric irritation, since if these are present they are generally aggravated by this class of remedies.

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[1] Belloc, who first introduced it, recommended that it should be made from the young shoots of the poplar, and stated that, given in doses of from 30 grains to 3 drachms in the twenty-four hours, it acted slightly as a laxative. Dr. Leared has of late praised the efficacy of the charcoal made from vegetable ivory for this purpose.

[2] Sydenham (Op. Omnia, Syd. Soc. Ed. p. 347) recommended their employment in hysterical or ataxic cases for a period of thirty days, and it is seldom that they can be taken with advantage for a longer period, though they may be resumed after an interval of ten days or a fortnight.

III.—Neuroses of the Stomach.

Synonyms.—*Erethism of Stomach* (Barras and Trousseau), *Gastralgia* (Barras, Valleix), *Morbid Sensibility of Stomach?* (Whytt, Johnson).

Definition.—An unnatural excitability of the stomach characterised by either extreme degrees of perversion of its functions, or by an excessive exaltation of its sensibility. Its principal symptoms consist of *pain, vomiting, perversions of its secretions,* and commonly of the *appetite.* These are frequently, but not constantly, associated with an impairment of the *digestive powers,* but are unattended by any appreciable anatomical change, and they usually occur under conditions capable of producing reflex irritation, or of inducing asthenic constitutional states associated with extreme nervous excitability.

The immediate mechanism of the production of such conditions appears to depend in some cases on nervous disturbance, which may be designated as primary, but in other instances they originate from the disorders of other, and sometimes of distant parts, by which the functions of the stomach are affected in the manner just described as reflex.

Etiology.—The predisposing causes of disturbances of the functions of the stomach resulting from perverted innervation, while embracing many of those general conditions which have been before enumerated as capable of inducing the atonic state, further include the special causes which are capable, in addition to weakness, of giving rise to extreme nervous excitability. We thus find that they are predominant in the female sex,[1] and occur with much less frequency among men. They are, however, rare before puberty,[2] and they are less common at advanced periods of life, than between the ages of fifteen and forty. A special proclivity at two different ages is, however, in the female sex determined by sexual conditions, the epochs of the appearance and cessation of the menstrual function being in them peculiarly liable to be marked by these disturbances; while in the male sex the only extraneous influences determined through age are such as result from the increasing anxieties and severer intellectual efforts which are often called for in those between thirty and fifty.

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[1] Georget, art. "Gastralgie," Dict. de Méd. x. 81, says that these nervous disorders are ten times more frequent in the female than in the male sex.

[2] With the exception of cases of vomiting from cerebral disease, and the rarer instances of simple uncomplicated reflex disturbance of the stomach arising during dentition, the periods of infancy and childhood are comparatively free from the severer forms of gastric disorder of a nervous character. The immunity thus enjoyed in the earlier ages of life is probably, in part at least, due to the absence of those profounder impressions made by moral emotions, which, as will be presently mentioned, serve in a great number of instances as their determining causes. Some cases are, however, recorded by Dr. Handfield Jones (Functional Nervous Disorders, pp. 412 et seq.), where neurotic abdominal pain was observed in boys before and at the period of puberty (see especially one by Dr. Martin, of Rochester, Brit. Med. Journ. July 16, 1859). In some, however, of these cases the pain appears to have been seated in the abdominal muscles rather than in the stomach.

Hereditary disposition is by some writers mentioned among their predisposing causes, and there seems no reason to doubt its occasional possible influence in this direction.

Of the special determining causes, *exhaustion* plays the most prominent part, and when combined with other depressing influences, and particularly with those of a moral character and operating through the nervous system, such as grief, fear, anxiety, or severe intellectual effort, it is an almost unfailing source of perversion of the functions of the stomach, which can only be referred to disturbed innervation. These, however, may originate under almost any circumstances of impaired vitality or of diminished constitutional power, whether apparently spontaneous, as in the states of anæmia and chlorosis (which though most common in the female sex have been observed to be thus associated in men), or which may be produced by hæmorrhage,[1] or by privation of food, by venereal excesses, and particularly by the habit of masturbation.[2]

Diseased states of the nervous centres may also act as causes of these perversions.[3] The chief facts which are known with respect to these relations have been already alluded to, and they do not thus far appear to require further description in this place.

By far the most frequent, however, of all the causes assignable as the starting-point of these disturbances, are the complex states expressed under the terms *Hysteria* and *Hypochondriasis*.[4] Their frequency in hysteria may be estimated by the statement of Briquet,[1] that of 358 cases of this affection, only 30 had no sign of "gastralgia," or "epigastralgia;" 130 had only pain at the epigastric region; while 187 had both pain and derangements of the digestive function: and this author states that the latter are among the first symptoms in females in whom hysteria is slowly developed.

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[1] Thus, Whytt (Works, p. 568) mentions a case of a young man who was bled largely for a pain in the side, arising from a fall from a horse. After some days he was attacked by a sense of intense cold in the stomach, and this was followed by intense pain recurring in a paroxysmal form two or three times in the twenty-four hours. The attacks continued for some weeks, but he gradually recovered under the use of "stomachics;" but having on another occasion experienced a similar, though smaller, loss of blood, the attacks of pain returned, but in a less severe degree. See also a case where pain and acid vomiting occurred after attacks of hæmorrhage from piles (Budd, p. 198). Hæmorrhoids in hysterical women are capable of producing extreme reflex disturbance of the stomach. In a case lately under my observation these ceased after the removal of the immediate exciting cause.

[2] Georget, Dict. de Méd. x. 84. Andral, Clin. Méd. ii. 193. Schmidtmann, "De Cardialgia," Summ. Obs. Med. vol. iii. p. 191, says—"Raro infantes puerosque invadit, crebrius juvenes et juvencas, atque eos in primis quando fœdæ deleteriæque sese addixere masturbationi: inde cardialgia in juvenibus obvia mihi semper suspicionem movet eos masturbari, atque disquisitione institutâ rarenter à vero aberravi." Ibid. iii. 205.

[3] Both gastric pain and vomiting may be excited by cerebral hæmorrhage, so as even to give rise to suspicions of poisoning. A case of this kind came within the author's own experience (recorded in Bennett's Clin. Med. 1865, p. 405). A similar one is quoted by Henoch from Krukenberg (Unterleibs-Krank. ii. 205). Empis, De la Granulie, p. 150, has recorded a case where epigastric pain and vomiting occurred in tubercular meningitis.

[4] The frequency with which the perversions of the moral and intellectual functions which are included under this title are found associated with all forms of dyspepsia may

Among other causes operating probably on the general or local nervous centres through changes in the composition of blood, may be mentioned malarial conditions, which may impart to neuroses of the stomach a specifically intermitting character.[2] From the present state of our knowledge of the pathology of gout, it would almost follow that this affection should be placed in the same category, though the dyspeptic disturbances occurring in patients of this class may be referable to widely different causes, among which inflammatory conditions probably play no inconsiderable part.

With them also may be included, but with a certain degree of reservation, the effects of long-continued alcoholic poisoning, which, as Dr. Budd[3] remarks, greatly resemble those produced by exhaustion.

Of the immediate exciting causes, sudden emotions, especially of a depressing kind, are among those most frequently cited as having given rise to these disorders; but a similar effect may be produced by moral affections of the same character acting through a longer period.[4] The influence exerted by painful emotions not only in arresting digestion, but in producing painful sensations in the epigastric region, is well known, and these effects are greatly heightened in the case of patients whose nervous susceptibility is more than usually prominent.

As special conditions, acting locally, may be mentioned the abuse of stimulants and condiments, and in particular the habit of taking large quantities of tea and coffee.[5]

Finally, in some cases the effects of idiosyncrasy must be considered.

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very properly give rise to the question, how far in all cases it can be regarded as standing to them in the relation of a cause, or whether in an almost equal number it is not either a direct consequence of the derangements of the digestion, or whether both these and the hypochondriasis are not together the expressions of a more general nervous disorder. There is, however, little doubt that, in whatever manner originating, the peculiar mental state accompanying this condition serves in no small measure to intensify the gastric disturbance already existing, through the exclusive direction of the mind to the sensations experienced in the stomach, the influence of which, as pointed out by Dr. Carpenter, in describing the effects of "expectant attention" on the organic functions, is by no means inconsiderable.

[1] Traité de l'Hysterie, p. 251.

[2] Niemeyer, loc. cit. p. 546.

[3] This is hardly, however, to be spoken of as a simple state, inasmuch as these cases are usually complicated by catarrh of the stomach, attributable, probably, to the immediate effects of the alcohol on the mucous membrane.

[4] See cases by Barras, Briquet, Andral.

[5] The latter agent is very commonly admitted to be an exceedingly frequent cause of gastrodynia, and of dyspepsia associated with much nervous disturbance. See Clarus, "Arzeneimittellehre," p. 666; also Wood, "Therapeutics and Pharmacology," i. 628. I have seen it stated, though I cannot find the original, that girls employed in needlework, and in the Manchester factories, have not unfrequently a habit of chewing tea, with results evidencing a greatly disturbed condition of innervation both of the general system and of the stomach. The habit does not, however, appear common in London.

Some of these have been already alluded to, and reference may be made to two cases by Andral,[1] in one of which the use of milk always caused violent pain, while in another honey invariably gave rise to vomiting.

The causes capable of acting on the stomach by reflex irritation, conducted from other parts, have also been dwelt upon in previous sections, and it is only necessary therefore briefly to recapitulate them in this place.

They are, as has been seen, very numerous, and include sympathies with parts whose apparent connexion with the stomach is very obscure, and some of which can only act, in all probability, through the general influence which *pain* exerts both on the appetite and on the digestive functions. Such are diseases of the external ear and of the teeth;[2] painful affections of the kidneys, testicles, and ovaries; disordered conditions of the lower portions of the alimentary canal, among which may be enumerated piles, worms, constipation, herniæ, including the omental and epiploic; diseases of the pancreas[3] and of the liver, and especially gall-stones and abscess of the latter organ; and with a frequency far exceeding that of all those just enumerated must be mentioned diseases of the uterus, including tumours, polypi, ulcerations, prolapsus, retro- and anteversions, and yet more commonly disturbances of menstruation, leucorrhœa, and the state of pregnancy. With the exception of the last-named state—which is, however, far more frequently associated with disorders of the stomach in weakly than in strong and healthy patients—there are few of the uterine derangements here enumerated which are not more or less associated with an impairment of the general nutrition. The majority also appear to be more truly connected either as cause or effect with the primary disorder, than to arise through the disordered digestion, which is frequently the last in the series; while, with respect to frequency, though not perhaps to severity, leucorrhœa and disordered menstruation hold the foremost rank among this class of etiological conditions.

SYMPTOMS.—The class of symptoms by which nervous affections of the stomach are especially characterised have been already alluded to as consisting chiefly of pain, vomiting, and certain forms of hypersecretion. These may, however, occur in every possible variety of combination with each other, and with other symptoms of indigestion, as well as in very varying degrees of severity. No special relationship can, as a rule, be traced between any of them and the different etiological circumstances just described under which they may occur; and causes apparently identical may, in different subjects, give rise to dissimilar symptoms; while, on the other hand, the most diverse perversions of function may occasionally be met with in cases whose mode of origin is to all appearance perfectly alike. Certain peculiar features do, however, mark some of the forms assumed by them, in connexion either with particular states of the system or with special causes of nervous irritation; and in the description about to follow, the disorders that occur in the course of hysteria, chlorosis, amenorrhœa, hypochondriasis, exhaustion, gout, and certain uterine disturbances, will be more specially considered; those originating from other reflex conditions, or from organic diseases of the cerebral centres, being comparatively so rare and exceptional, that though their mode of causation is occasionally of diagnostic importance, they can hardly be included in a categorical description of the symptoms of the more usual forms.

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[1] Pathologie Interne, i. 153.

[2] See a case by Liederer of a young lady in whom a false tooth fixed to the socket of a diseased one caused regularly returning attacks of pain and vomiting, which ceased immediately on removal of the pressure from the dental nerve. Allg. Weiner Med. Zeit. No. 24, 1861.

[3] Claessen, quoted by Bamberger.

The modes of invasion of these symptoms of nervous "erethism" may be either gradual or sudden, standing in this respect in an almost direct relationship to conditions similar in point of time or intensity of the exciting cause.

In the class of hysterical cases, *loss or depravation of appetite* is sometimes an early symptom, proceeding in many cases either to an absolute anorexia, which may even involve the nutrition of the patient in considerable danger;[1] or to some of the extraordinary perversions known under the names of Pica or Boulimia.[2] Sometimes the last-named symptom becomes predominant, and the patients are affected with a ravenous and almost insatiable desire for food, compelling them to eat at all hours of the day and night, and the origin of which in perverted sensation is evidenced by the feelings of sickness, pain, and faintness which ensue if the gratification of this desire is withheld.[3] "The psychical relations of the sense of hunger are altered; there is no appetite, and taking food affords no enjoyment."[4]

Severer symptoms, however, often speedily ensue, and among the most prominent of these is *pain*, which, however, varies greatly in duration and intensity. Commencing ordinarily with a sense of constriction or oppression, or sometimes with a feeling of distension or weight, it is followed by sensations of an agonizing character, which are variously described by the patients as consisting of heat, cold, tearing, gnawing, rending, or twisting. In the severer attacks the heart's action is interfered with, becoming fluttering and irregular; the extremities are cold, and there is a tendency to syncope: in some cases convulsions are said to have ensued from the severity of the pain (Schmidtmann). Pain of this nature, though often aggravated by slight pressure upon the abdominal muscles ("épigastralgie" of Briquet), is usually relieved when it is made more firmly and persistently; and this is especially true of those forms that are associated with cramp or flatulence, but the conditions observed in this respect are somewhat uncertain.

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[1] Briquet, p. 254.

[2] Pica, Citta, Malakia Pseudorexia, are terms signifying a perverted appetite, in which desires are experienced for unnatural kinds of food—coal, chalk, pins, or even loathsome substances. Boulimia is applied to an excessive desire for food. Copland's Dict. of Med., art. "Appetite." Landre Beauvais, Dict. Sciences Méd. iii. art. "Boulimia."

[3] I have seen this state in a most intense form in an elderly single lady of between 70 and 80 years of age, who certainly has never exhibited any signs of hysteria. Every cause depressing her health is followed by a return of the symptom. The hunger distresses her chiefly at night, when she is obliged to eat several platesful of sandwiches. No other disease is present. She suffers from indigestion, but not so much when the hunger is present. She has been temporarily relieved by bromide of potassium, nitrate of silver, strychnia, and arsenic. The latter procured the longest period of immunity; but the symptom repeatedly returns whenever any impairment of health takes place. The condition, though generally occurring in the female sex, may sometimes be met with in males, as is shown by an extremely marked case recorded by Chomel, Des Dyspepsies, p. 94.

[4] Romberg, loc. cit. p. 107. Romberg remarks that this hyperæsthesia rarely occurs in an isolated or idiopathic form, and that it is found in a variety of morbid states—sometimes appearing as reflex or sympathetic, sometimes associated with other hyperæsthesiæ—and is observed during convalescence from severe diseases, in hysteria, helminthiasis, ergotism, or as the precursor of other diseases, and especially of gout.

The pain is sometimes associated with distension of the abdomen; at others this is sunk and retracted (Romberg). It is essentially paroxysmal in its character, returning sometimes at irregular, in other cases at regular periods on successive days in cases where the attacks are not attributable to malarious influences. In other cases, it returns only at the menstrual periods, and there is comparative immunity in the intervals.[1]

The duration of the attacks is variable. They may last only for a few minutes, or may continue for hours. They frequently terminate in gaseous eructation, or with the ejection of a watery fluid, which may be acid, or alkaline (pyrosis), or of mucus of an alkaline character, and of variable degrees of tenacity; and they are commonly followed by a sense of soreness at the epigastrium, and by great exhaustion.

The relation of the effects of the ingestion of food to the pain is subject to some variations. There is one distinct class, before alluded to, where this gives distinct relief;[2] and these cases form not only the larger number, but are those in which there is least suspicion of the accuracy of the diagnosis. This effect is certainly that most commonly met with in cases where the gastrodynia is associated with depressed vitality arising from exhaustion, or with nervous irritability dependent on moral or emotional causes. Another remarkable feature in these cases is, that insipid food and demulcents often cause much more pain than substances which are not only less digestible, but are even irritating in their character; and this peculiarity, which has been observed by numerous writers,[3] is not without value in diagnosis.

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[1] Barras gives two cases where gastrodynia was associated with suppression of the menses: in one of these the pain was less violent when the discharge became more abundant. In this latter case there was also flatulence, and the pain was relieved by food. (Loc. cit. i. 587.) Niemeyer, loc. cit. p. 545, also gives a case of this character, where the pain only occurred at the menstrual period, but in which the application of leeches to the cervix uteri instantly caused its return. It must be remembered, however, that an increase in the severity of the pain at the period of menstruation has been observed in some cases of gastric ulcer.

[2] See Budd, loc. cit. pp. 282, 283.

[3] "An uncommon delicacy of the nerves of the stomach, which may be either in a great measure natural or brought on by disease, ... excessive grief, or other causes,

In other and rarer instances, which especially occur in aggravated forms of hysteria, and in some where the pain in the stomach is of reflex origin, and more particularly where it is due to uterine disturbance, the ingestion of food is immediately followed by severe pain, which is only relieved by vomiting; and though in some cases the appetite may be preserved, the fear of the agony caused by the food entering the stomach prevents the patient from eating.[1] In other instances, pain of this character is only felt some hours after food; but here the conditions are more complex, and pyrosis or great distension of the stomach from flatus is often present; in which latter case, though much of the pain felt may be due to the spasmodic contraction of its coats, some is also, in all probability, to be attributed to the cramp-like action of the abdominal muscles.

The state of the digestion sometimes affords a valuable criterion of the nervous origin of these affections; in many cases it is entirely unaffected in the intervals of the attacks,[2] while in others the ordinary symptoms of atonic dyspepsia are present. The tongue is, as a rule, clean, though often pale, broad, and flabby:[3] the bowels are generally confined, but the fæces are not otherwise altered. In cases, however, to be hereafter alluded to, when nervous excitability co-exists with various degrees of inflammatory irritation of the stomach, these signs are lost. One remarkable feature with regard to these attacks is, that where any predisposition to them is present they may be brought on by painful moral emotions.[4]

Vomiting, though often attending the paroxysmal attacks last described, may also occur as an independent condition, unaccompanied by pain. This, though common in cases where it originates in reflex disturbances, or in certain disorders of the central nervous system, may also be observed in states characterised by simple perversion of function, and especially in hysteria. Its isolated appearance in the latter class, independently of other disturbances of sensation, is, however, the exception rather than the rule, though in some instances

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is to be distinguished from that . . . . increased sensibility, which is the consequence of an inflammation, or of an aphthous state of those parts, since in these last cases every acid substance gives them pain, whereas in the former many insipid and seemingly innocent substances produce great uneasiness in the stomach and bowels, while volatile spirits, strong wines, brandy, and spiceries, are not only inoffensive, but often necessary for allaying those disorders which are produced in the first passages by such causes as would scarcely produce any disturbance in the second state."—Whytt, loc. cit. p. 544. See also the same author, loc. cit. p. 566; see also Barras, loc. cit. i. 35, 414, 440.

[1] Briquet, p. 256. These cases are, however, those in which the accuracy of the diagnosis from ulcer must often be held in doubt, even when made by the most competent observer.

[2] This can only be said to be relatively true in regard to the diagnosis of these affections from those of an inflammatory nature. The same fact is often observed in the progress of cancer, and sometimes of ulcer, when associated with epigastric pain.

[3] The varieties of appearances of the tongue described by Dr. Todd, loc. cit. p. 632, as indicating this state, viz. "a thin white gauze," "a milky appearance," or "a covering of frothy mucus," belong, I believe, to catarrhal conditions.

[4] Whytt, loc. cit. p. 560.

such pain belongs more to the class which Briquet has termed "épigastralgie," than truly referable to the stomach.[1]

Nausea usually precedes to some degree the expulsory act; but its duration and intensity are shorter and less marked than in the vomiting which attends inflammatory or organic diseases of the stomach—approximating in this respect to the conditions observed in the vomiting from cerebral causes.

In some cases intervals of several hours, or even days, may elapse between the recurrences of this symptom, and during them the digestion may be good, or there may be (as in pregnancy) some degree of acidity, apparently from hypersecretion, after each meal; but in others, and apparently in proportion to the severity of the exciting cause, and also in pregnancy and hysteria, the vomiting tends to become continuous. Under such circumstances the food is rejected either as soon as taken, or within a few hours after eating, being often returned completely undigested, and rarely associated with bile or mucus (Briquet). Even when vomiting is severe, the appetite may in some hysterical cases continue, and may even present an apparent increase of intensity, so that the patient's time may be passed in alternate vomiting and eating;[2] but in other instances, when the disorder has commenced after moral depression, and when pain is also present, there may be a more or less absolute anorexia.[3]

In many cases of hysterical vomiting it has been a matter of repeated observation that the general nutrition and strength remain but little affected; but when the vomiting is severe and constant, emaciation may rapidly ensue from the loss of food thus occasioned. The occasional dangerous effects of this symptom in pregnancy are well known, and have been already alluded to; and others of a similar character are detailed by Andral[4] and Budd,[5] where, although vomiting had continued long, the stomach was found healthy after death. In some of the cases of the latter class, though fever was absent in their earlier periods (thus conforming to the rule generally observed), it appeared, associated with delirium, towards their close, and in some instances assumed a distinctly hectic character.

The effects of varieties of food in promoting or retarding the occurrence of vomiting are almost as variable as are their relations to the symptom of pain. In the severer forms just alluded to, solids and meat have been known to increase both the pain and the vomiting (Budd); but when hysterical symptoms are well marked, indigestible and apparently unsuitable food may be tolerated when ordinary aliments are rejected as soon as taken.[1]

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[1] See a case related by Briquet, loc. cit. p. 218, of a girl who, after severe moral emotion, was brought to the hospital with violent pain at the epigastrium, together with vomiting, which excited the suspicion of poisoning. The pain was relieved in ten minutes by faradisation.

[2] Briquet, loc. cit. p. 255.

[3] Andral, loc. cit.

[4] Clin. Méd. ii. 175—179.

[5] Loc. cit. p. 261. With regard to Andral's cases, however, the conclusions drawn from the apparently healthy state of the stomach should be received, in the author's opinion, with some caution, on the grounds stated in the chapter on the post-mortem appearances presented by the stomach.

There is another form of dyspepsia occurring under nervous influences, of which I have seen some examples, but which, as far as I am aware, has been fully described only by M. Trousseau.[2] It occurs both in males and females, under conditions usually preceded by some cause of exhaustion, especially affecting the nervous system; and my friend and colleague, Dr. Ringer, informs me that he has also observed it in children, and I have also met with it under five years of age. It is attended with an excessive appetite (boulimia), and by a sense of want following too speedily after food has been taken. The characteristic symptom is, however, that diarrhœa is present, and the evacuations, which are usually preceded by borborygmi and colicky pains, are induced with great facility by slight causes of an emotional character, and they contain considerable quantities of food which have passed undigested through the intestinal canal. Frequently an evacuation occurs immediately on rising in the morning, and is followed by a great sense of exhaustion; and I have observed in some cases that this can be delayed until the usual hour after breakfast, by simply taking a small quantity of food before rising. Trousseau ascribes these symptoms to an undue irritability of the muscular coat of the stomach and intestines, hurrying the food taken too rapidly through the whole canal, without permitting time for its due elaboration; and the influence of appropriate treatment strongly corroborates his opinion.

The affections of the general system frequently associated with the conditions which have now been described are nearly all such as point to their relation to a common cause. Many of them belong to the category of neuralgias, which occur either in distant or in proximate parts, and their appearance often alternates with remissions in the gastric disturbances. These often affect the dorsal, thoracic, abdominal, and intercostal muscles; but they may assume the forms of toothache,[3] hemicrania, lumbago,[4] or sciatica.[5] Instead of pain, other symptoms of nervous excitability may be present, such as attacks of cardiac palpitation,[6] or of strong pulsations of the abdominal aorta,[7] which often occur both during and in the intervals between the attacks of abdominal pain, and are distinguished by their sudden invasion and cessation, and by their rapid exacerbations. Dyspnœa,[1] globus, hiccough, paralytic,[2] syncopal,[3] or convulsive affections, salivation, and a copious discharge of pale urine[4] have all been observed, with greater or less frequency, as concomitants of these affections of the stomach, and indicate very clearly the condition of perverted innervation from which its disorders arise, and which can be traced to the states of chlorosis, hysteria, melancholia,[5] or hypochondriasis, in which they take their origin.

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[1] See a case by Barras, loc. cit. i. 496, where the first food retained was a salad made with hard-boiled eggs. A precisely similar one is recorded by Briquet, loc. cit. p. 307; another by Guipon, loc. cit. p. 349, when the acid vomiting of pregnancy was immediately arrested by the use of *beer*. Valleix, Bull. Thérap. Oct. 1849, has noticed that meat and champagne were retained when milk was rejected, and that a kind of food which was retained one day was vomited on the next, and *vice versâ*.

[2] Clin. Médicale, ed. 1862, pp. 354, 365, 428, 429. This is probably also allied to the affection alluded to by Abercrombie, "Diseases of Stomach and Intestines," 3d edition, p. 71, and by Whytt, loc. cit. p. 530, as *quick digestion*.

[3] Common in pregnancy.

[4] See a case of lumbago alternating with gastrodynia, Andral, Clin. Méd. ii. 297.

[5] Abercrombie, loc. cit. p. 86. Barras (i. 440) gives a case where a patient who had been subject to periodical headache became attacked with intermitting gastrodynia accompanied by the vomiting of mucus, but not of food, and which ceased on the supervention of sciatica. Two or three such attacks, with a similar order of sequence, were repeated at considerable intervals.

[6] This, however, is a symptom common to all forms of dyspepsia.

[7] Barras, loc. cit. p. 411. Walshe, Dis. of Heart, 3d ed. 438. Lebert, Virchow's Handbuch, V. ii. 53.

The sleep also is affected in the majority of cases, and particularly in hysterical patients, who not only experience a difficulty in going to sleep, but are liable to awake during the night, with a great sense of exhaustion and hunger. Insomnolence is also commonly observed in patients of both sexes in whom disorder of the stomach has been caused by anxiety or over-fatigue, though in them the feeling of hunger on waking is usually wanting. In other instances, however, of the latter class the sleep may be heavy, but it is often unrefreshing and disturbed by dreams.

Many of the disturbances of the functions of other organs of the body have been already spoken of in connexion with the description of special symptoms, and it is unnecessary again to repeat the remarks made on the subject of the appetite, the digestion, or the constipation and flatulence which, with some exceptions, mark these cases, nor on the irregular and scanty menstruation, or the leucorrhœa attendant on anæmia or chlorosis, whose influence has been already largely insisted upon. It has been observed that in many cases where hypochondriasis is most marked, oxaluria has been present. This symptom is however common to a great number of diseases, and in some cases it probably depends more or less directly either on an excess of animal food (Niemeyer)[6] or on imperfect assimilation, when its occurrence is rather to be associated with dyspeptic disturbances of another character, where the hypochondriasis, instead of being primary, is secondary to the disorder of the digestion, than with the more purely nervous disturbances now under consideration. In others, however, it appears to be directly associated with nervous disturbances of this class.

One important variety, to which Whytt first called attention, is that where severe disorder of the stomach occurs in connexion with the gouty diathesis, and ceases with the appearance of the disease in one of the joints.[7] The attacks, however, as Whytt further remarked, are less liable to affect robust and strong individuals in whom the gouty affection is regularly developed, than in those of "weak fibres and very sensible nerves," who have "rarely any disorder like true gout," but who are liable to "craving or faintness, nausea or vomiting, flatulent swellings, borborygmi, low spirits, cramps, convulsive and violent pains in the stomach and bowels, and an increased secretion of saliva." In other cases severe spasmodic affections of the stomach occur in the course of gouty attacks, and are characterised by violent abdominal pain, intense distension from flatus, and severe sinking, followed in some instances, when wine has been drunk, by acid vomiting, which has given relief.[1] The nature of these disorders is, however, a matter of some doubt; and though evidence regarding them is still wanting, there is considerable probability that many of them are not simple neuroses, but are rather to be classed under the category of acute indigestions, complicated by a predominance of nervous excitability together with prostration.

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[1] Whytt, loc. cit. p. 560.

[2] A very interesting case of this nature, by Dr. Perceval, is to be found in the Med.-Chir. Trans. iv. p. 17.

[3] Guipon, loc. cit. p. 118.

[4] For a case of this kind occurring in a *male* patient, see Whytt, loc. cit. p. 557.

[5] See Marshall Hall, On the Mimoses.

[6] Loc. cit. i. 554.

[7] Works, pp. 556, 559.

Prognosis.—The course of these affections is, as may be inferred from their history, very variable. Their duration depends in a large measure on the persistence of the exciting cause, ceasing with its cure, as is often observed in cases originating in anæmia or chlorosis, or in those arising from reflected irritation, especially when the cause is seated in the uterus.[2] The rapidity of their disappearance on the supervention of some other nervous phenomena affecting other parts, or of an attack of gout in cases when this diathesis is present, has already been a subject of remark. Barras[3] has observed that in some cases they disappear on the supervention of acute diseases, as fevers,[4] pneumonia, abscess, or variola, but are liable to reappear during or after convalescence.

The majority may continue for years with but little danger to life; and this is true not only of the minor degrees of hypochondriacal uneasiness, but even of some of the severer cases of hysterical pain and vomiting. The hysterical forms, like all the other phases of this malady, are liable to diminish in severity and even to disappear with advancing life.[5]

Briquet remarks that the vomiting of hysteria, though sometimes very obstinate, is the least dangerous of these affections. That it may, however, be attended with fatal consequences, and especially when constantly ensuing after everything eaten, and when associated with severe pain, is evident not only from his, but also from observations recorded by Andral, and Barras, and Budd. The danger resulting to the general nutrition from the more absolute forms of anorexia has been already remarked upon.[1]

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[1] See a case by Sir C. Scudamore, quoted by Dr. Garrod, "Gout," p. 503.

[2] An interesting case of this kind is quoted by Henoch, from the third edition of Romberg's work. Henoch says that in some cases of cardialgia the pain has been known to occur only at the menstrual periods, *to cease with pregnancy, and to return after delivery.*

[3] Loc. cit. i. 539.

[4] Τὰ σπασμώδεα καὶ τετανώδεα πυρετὸς ἐπιγενόμενος λύει. (Hippoc. Prænotiones, Works, Kühn's Ed. i. 289.)

[5] "Progrediente ætate cardialgia crebro sponte evanescit; multas novi mulieres, quæ tempore juventutis et ætate florente frequentissime hoc malo macerabantur, ad senium perventas ab eo omnino immunes viventes." (Schmidtmann, loc. cit. iii. 207.)

The uncontrollable vomiting of pregnancy may also at times assume a very dangerous character. Thus, of fifty-eight cases collected by Cartaya,[2] thirty were fatal, and twenty-eight were cured after abortion or the death of the fœtus—fourteen of the latter having had premature delivery induced with success, while one recovered after the application of leeches to the os uteri, and two through the use of champagne.

THE PATHOLOGY of the functional disturbances of the stomach, of which the description has here been given, is involved in the obscurity which attends that of a large class of nervous affections, and of which, indeed, it may be said, that our knowledge consists rather of isolated facts than of comprehensive inductions. Many of the leading phenomena, attesting the influence of the nervous system in the production of the symptoms here alluded to, have been already considered, and the author has but little to add to what has been before stated, except to pass briefly in review some of the better known conditions of nervous disturbance which are applicable (in many cases, however, only inductively) to these affections.

It is well known that excessive excitability of the nervous system stands in an almost inverse relationship both to the due co-ordination and to the efficiency of the functions under its control, and that conditions of weakness or mal-nutrition of the nervous centres are evidenced by perversions in the harmony of their action characterised by an apparent excess of activity in one direction, but attended by deficiency in another.[3]

Both the sensory nerves, and also those supplying both the voluntary and involuntary muscles (including among the latter the contractile coats of the blood-vessels), are subject to similar perversions of action. Thus it has often been noticed that when any part is the seat of severe pain, its power of distinguishing sensations is proportionably impaired, and local hyperæmia very frequently ensues. Hyperæsthesia is also occasionally combined with muscular paralysis or with convulsive movements, as may be sometimes observed in neuralgias of the fifth nerve.

The experiments of Lister and others have shown that the actions of the visceral and vaso-motor nerves are exalted by impressions of moderate intensity made on the nervous centres, but are paralysed when these are carried to extreme degrees of stimulation. It is further highly probable that these impressions can be equally produced by peripheral stimulation of the sensory nerves as by direct excitation of the central roots of the motor fibres; and, therefore, the reflex effect resulting from the former will depend not only on the sensibility of the peripheral branches, but also on the degree of excitability of the nervous centres. Hence when this excitability is excessive, the effects of a moderate peripheral stimulus will resemble those produced artificially by excessive direct stimulation in the healthy state, and will give rise to paralysis or convulsion according to the proportion borne between the amount of stimulus and of the excitability present. Dr. Handfield Jones[1] has also adduced some reasons for believing that vaso-motor paralysis, determining excessive secretion, may co-exist with undue irritability of other nervous branches, since excessive secretion from the eye and nose are known to be frequently attended with hyperæsthesia of the sensory nerve of these parts. If this view be correct, it is comparatively easy on the data above given to frame an hypothesis, with relation to these affections of the stomach, explanatory of the phenomena of hyperæsthesia associated with secretions either excessive in amount or perverted in quality, and also with spasmodic movements indicated by vomiting, and occurring in conditions signalized by morbid excitability of the nervous centres. The cases also in which reflected nervous action is observed to proceed in unusual, and probably in abnormal directions, appear to be explicable by the same data, and are further illustrated by some striking examples of such occurrences in parts more directly accessible to observation.[2]

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[1] Of course this must not be confounded with those simulated forms of anorexia where the patients eat by stealth.

[2] Schmidt's Jahrbücher, 1855, iv. 60.

[3] Thus (to cite familiar instances) a muscle in violent convulsion is unable to execute voluntary movement; a weak eye is conscious to a morbid degree of the impression of light, while its power of distinguishing objects is diminished; mental excitement, attended with rapid thought and vivid imagination, is generally deficient either in perspicacity of ideas or in the power of sustained attention or of logical precision.

The effects of anæmia as a cause of local pain, to which some allusion has been made by recent writers, and especially by Dr. Anstie, may possibly serve in some cases to explain the sensation of pain felt when the stomach is empty, and also its relief by food, which causes an increased afflux of blood to the part; and it is not without its parallel in the neuralgias of other parts, which are cured by a blister placed on the course of the nerve, or directly on the seat of pain.[3]

Of any special anatomical conditions discoverable in these cases we are as yet in ignorance; in fact, the absence of these constitutes their distinguishing feature, as contrasted with those cases where such lesions are found to explain the phenomena observed during life.

The question regarding the possibility of organic disease resulting as a consequence from long-continued disturbances of function, excited through the nervous system, is of very considerable importance, but distinct clinical evidence of any anatomical lesion of the *stomach* having originated independently from this cause is very defective, and the general question is of too wide an extent to permit of its appropriate discussion in this place.

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[1] Functional Nervous Disorders.

[2] See an interesting case cited by Dr. H. Jones, "Functional Nervous Disorders," p. 11, of paralysis of the muscles of the eye, attended with circumorbital pain, resulting from necrosis of one of the phalangeal bones of the hand.

[3] Anstie, Stimulants and Narcotics, p. 214.

DIAGNOSIS.—The diagnosis of the several forms of nervous disturbance from those arising from the organic diseases, the symptoms of which they more especially simulate, is often a matter of extreme difficulty.

(*a*) The chief criteria upon which reliance must be placed, are the recognition of a constitutional state predisposing to nervous excitability, and particularly the presence of conditions of great exhaustion or the hysterical diathesis.

(*b*) The presence of causes of sympathetic irritation, and the aggravation or alleviation of the symptoms of the stomach affection in a direct ratio with an increase or diminution of the severity of these.

(*c*) The disproportion observed between the severity of the gastric symptoms and the general state of the patient, and particularly the absence of emaciation when severe vomiting forms a prominent feature. The evidence from cachexia is of less value, since anæmia is frequently associated with nervous disturbance; and chlorotic females, whose menstruation is disordered, are frequently the subjects of ulceration of the stomach.

(*d*) In the case of pain, its frequent complete remissions are almost diagnostic of the absence of inflammation, though by no means excluding the possibility of its cause being due to either ulcer or cancer. Pain from inflammatory affections, unless due to corrosive poisons, seldom, however, presents the intensity of suffering produced by neuralgias of the stomach. The seat of the pain, and especially of tenderness, requires careful investigation; and when this is exclusively superficial,[1] it would strongly favour the opinion of the nervous character of the disorder, even when under these circumstances it is allied with vomiting.[2]

(*e*) Pain occurring when the stomach is empty, and relieved by food,[3] is almost distinctive of its nervous origin. Exceptional cases of this nature have indeed been observed in ulcer and cancer, but they are of great rarity. The influence of the nature of the food upon the pain, and also upon vomiting, is another criterion, which is not without value. It has, however, been occasionally noticed in exceptional cases of ulcer, that stimulants have given relief.

(*f*) The co-existence of other neuralgiæ, and the alternation of pain with these, are also a strong ground for suspicion that the affection of the stomach belongs to the same class. Intercostal neuralgia is, however, of minor value as a means of distinction, as it may be observed in some cases of ulceration. Dorsal pain is common in both classes, and affords but few positive features of distinction between them. Spinal tenderness, though commonly present in cases of neuralgia, is occasionally absent, while on the other hand it may exist in cases of ulceration. The presence of other neuroses is also a valuable aid, though it must be remembered that in some females excessive sensibility may co-exist with an inflammatory condition of mucous membrane, and that in such cases the recognition of the latter is of great importance in relation to treatment.

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[1] I have known, in some cases where, from the presence of hæmatemesis and of severe pain occurring immediately after food, there was the strongest suspicion of the existence of ulceration, there was yet great superficial tenderness. Careful examination, however, revealed the existence of tenderness on deeper pressure in limited portions of the epigastric region.

[2] See Briquet's cases, before quoted.

[3] A craving for food is sometimes experienced in cases of chronic catarrh, and also of ulcer; but when yielded to, it usually aggravates the uneasiness. There are, however, exceptional cases of an hysterical character, before alluded to, where, though a strong desire for food exists, it is speedily followed by vomiting.

(*g*) Vomiting from nervous disturbance often resembles that arising from cerebral causes in the facility with which the act takes place, and particularly in the absence of relief to the pain or nausea which attends the evacuation of matter irritating a stomach which is the subject of organic disease. The co-existence of pyrosis with pain affords but little conclusive evidence as to its origin, since this symptom may arise from causes both of organic and of purely functional nature, the character of which must be determined by the attendant symptoms.

(*h*) The absence of pyrexia usually distinguishes these affections from those of inflammatory origin, though it is of no value in their diagnosis from ulcer and cancer, in which disorders a febrile state is very seldom observed except from the presence of some other inflammation capable of exciting it.

(*i*) The state of the tongue usually serves as an additional guide in the diagnosis from inflammatory affections. It is usually unaffected when the disturbance is of nervous origin; its characters in the catarrhal states have been already described.

On the other hand, the tongue is of little value in the distinction of the neuroses of the stomach from ulcer and cancer, where the appearances which it may present are very uncertain, and depend more on the presence or absence of the complication of catarrhal states than on any other cause.

(*k*) The diagnosis of severe attacks of spasm in gouty cases from those of inflammatory origin is one of considerable obscurity, as the intense depression often masks to some degree the inflammatory symptoms. The chief symptoms which should create suspicions are febrile disturbance, a loaded tongue, tenderness on pressure at the epigastric region, and burning or heat at the stomach, as distinguished from the cramp-like pain which usually marks the attacks of a more spasmodic form.

THE TREATMENT of the various forms of nervous disturbance of the stomach may be briefly summarized as consisting in modifications of the tonic and stimulant plan already recommended for atonic dyspepsia; and their cure under this system affords further and valuable evidence of their true nature.

The discovery and cure of any source of peripheric irritation from which the symptoms may originate is of the very first importance. It does not, however, seem necessary to do more than to remind the reader of the frequency with which in the female sex these diseases have their starting-point in uterine disorders. It will, however, conduce somewhat to perspicacity if the remedies which may be more particularly directed to the condition of the stomach are mentioned in relation to the symptoms for which they have been found most efficacious.

In the relief of neuralgic pain iron holds a prominent place.[1] In the majority of instances the neutral preparations, and especially the carbonate, are both the most serviceable and also the safest. The dose should not exceed five grains, since larger ones often provoke colic and intestinal disturbance (Trousseau). Some writers, however, as Abercrombie, Dr. Handfield Jones, and Henoch, recommend the sulphate in doses of one to two grains. Henoch advises that it should be used in combination with morphia, and Abercrombie with aloes and pulv. aromat. taken twice daily, and the latter formula will at times be found serviceable when constipation is present.

Arsenic has been found very useful by Dr. Leared[2] in cases of severe neuralgic pains when there are no signs of irritability of the stomach, as evidenced by redness of the tongue, and pain following immediately after the ingestion of food. When this symptom has however appeared in phthisis, Dr. Leared has also found arsenic useful. The cases where it is most indicated are those when the neuralgic condition has followed mental or physical exhaustion, and in those of malarial origin.

Nux vomica is very useful in these cases, as also in some forms of pyrosis.

Bismuth and the nitrate and oxide of silver are also valuable. Hydrocyanic acid has appeared to the author of inferior efficacy in relieving either pain or vomiting of nervous origin.

The utility of opium in painful affections of the stomach can scarcely be overrated, though the usual caution is necessary with respect to its habitual use. A single dose will often permanently relieve pain of many days' standing, and its value has been strongly insisted upon by most writers on these disorders.

It is of special use in gastrodynia arising from anxiety and exhaustion, but its value is not inconsiderable in many hysterical cases; and it has been remarked that, when it agrees, its effects in producing constipation are scarcely perceptible.

In pyrosis, bismuth combined with opium seldom fails to relieve; though, in order to complete a cure, a more direct astringent is sometimes necessary, and for this purpose the compound kino powder is the best remedy that can be employed. Pain from flatulence is in these cases best treated by ether or by aromatic spirit of ammonia, and the former agent is sometimes useful in checking hysterical vomiting. Trousseau[3] speaks highly of the use of valerian and assafœtida in the wearing uneasiness which he terms "anxiété épigastrique."

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[1] See especially Andral, Clin. Méd. ii. 223, and Dr. Martin's case, before alluded to.

[2] Brit. Med. Journ. 1867, vol. ii.

[3] Traité de Thérap. ii. 307.

In the general irritability of the nervous system, associated with dyspeptic symptoms and oxaluria, which occur among the effects of anxiety or exhaustion, the sulphate of zinc, first recommended by Dr. Golding Bird,[1] and the oxide, subsequently used by Dr. Marcet[2] in cases where the same symptoms have followed the use of alcohol, and even in some where the latter cause has not existed, often prove of considerable service. They both seem to act as "tonics" in this condition, and the oxide has the additional advantage, if given at bedtime in doses of from two to three grains, of procuring sleep.

When the pain is very severe, relief has sometimes been experienced from the application of plasters of belladonna or of opium to the epigastrium, or from counter-irritation in this region by means of croton oil or blisters, and the latter may be followed by the endermic application of morphia.[3]

In obstinate cases it may be well to try Briquet's recommendation of faradisation.

Vomiting is sometimes a very difficult symptom to overcome, and the possibility of its fatal termination has been already alluded to. When very severe it is important to give the stomach rest by the employment, during a certain period, of nutritive enemata, a plan which should never be omitted when vomiting, from whatever cause, is severe and obstinate. The value of rest is further illustrated by a case quoted by Sir T. Watson and other writers from William Hunter,[4] where a boy, reduced to an extreme degree of emaciation by constant vomiting, attended by severe pain, was fed by spoonfuls only of milk frequently repeated, with the effect of completely arresting the vomiting, and enabling the stomach to bear more substantial food, the result being that the patient was completely cured; and numerous cases since recorded have confirmed the advantages of this method.

Opium is often of considerable value, and I have known a few doses of three or four drops of laudanum speedily check vomiting resulting from disease of the uterus, which had persisted for some weeks previously. It may be given an hour before meals, and in severe cases its administration by enemata may often be resorted to with advantage.

Iced effervescent drinks, combining the effects of cold with the sedative[5] effects of the carbonic acid, also prove useful; and benefit is sometimes experienced from the use of champagne, and also from effervescent draughts containing hydrocyanic acid.

Bismuth, as has been remarked by other writers, is of comparatively little service when vomiting is purely sympathetic, and particularly when it arises from disorder of the uterus; but when any inflammatory state is present and complicates the nervous disturbance, it may often be employed with advantage combined with hydrocyanic acid.

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[1] Loc. cit. p. 256. [2] Chronic Alcoholic Intoxication, p. 106.

[3] It is probable that in many of the cases where the efficacy of such remedies has been recorded, the pain so relieved has more truly been seated in the abdominal muscles, where, as before remarked, its presence may complicate the gastric pain. In some of these cases the hypodermic injection of one-sixth of a grain of morphia over the seat of tenderness has afforded relief, and I have known this plan to be most efficacious in the severe pain associated with a cancer of the stomach which had formed adhesions to the anterior abdominal wall.

[4] Med. Obs. and Enquiries, vol. v.

[5] Pereira, Mat. Med. i. 125. Sir J. Simpson's Obstetric Works, ii. 769.

The oxalate of cerium (first introduced into practice by Sir J. Simpson,[1] of Edinburgh) has been found very efficacious in some cases of vomiting in pregnancy. It should be given in pills in doses of one or two grains.

In many of the milder cases of acid vomiting or troublesome heartburn of pregnancy, hydrochloric acid combined with tincture of nux vomica, given before meals, often proves of service. I have seen less benefit result from the use of the acid alone; and part of the efficaciousness of the remedy is in my opinion due to the influence of the nux vomica.

Pepsine is also occasionally of value in these affections, though the mode of its operation is not very intelligible; but the administration of one or two doses is sometimes sufficient to enable the food to be retained, and the habit of rejecting it, being once broken, seems in some cases to be the essential feature of the cure.[2]

Among other remedies that have been found efficacious in hysterical vomiting are the douche, shower-bath, or cold affusion to the body and abdomen.[3]

In the vomiting of pregnancy, Bretonneau reports that he has found great service from frictions of belladonna over the hypogastric region;[4] and the applications of the extract to the cervix uteri has been made with the same intention by Cayeaux[5] with good effect.

Acupuncture has also in some cases been efficacious,[6] and in others faradisation[7] has been found of value.

The internal administration of tincture of iodine has been known to check the vomiting of pregnancy,[8] but it does not appear to have been tried in this country, and I have no experience of its efficacy.

Finally, it must be recollected that troublesome vomiting apparently of the same kind may sometimes be caused by constipation, and that the use of aperients may be absolutely necessary to overcome it. In these cases the mixture of carbonate and sulphate of magnesia in doses of a scruple of the former to two drachms of the latter in some aromatic infusion should be administered repeatedly every four or six hours, or castor oil may be taken on several successive mornings until the bowels have acted freely.

The foregoing list, though affording a great variety of choice, indicates also that there are probably concealed differences in the nature of the affections for which they are found useful, which are not as yet fully elucidated. The more ordinary kinds of neuralgic pain, when independent of causes of reflex origin, are as a rule easily controlled by iron, nux vomica, opium, ether, nitrate or oxide of silver, hydrocyanic acid, bismuth, or arsenic; but it is in the cases of severe hysterical pain and vomiting, or where these symptoms originate from causes of irritation in distant organs, that the greatest difficulty is experienced, and in these there is a considerable degree of uncertainty in the effects of treatment.

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[1] Sir J. Simpson's Obstetric Works, i. 313.

[2] This point has been ably insisted upon by Dr. Chambers.

[3] Andral, Clin. Méd. ii. 196. Barras, i. 480, quotes a case from L. Frank, where a lady, who had vomited her food for eight years, retained it when taken in a bath.

[4] Trousseau and Pidoux, Traité de Thérap. ii. 76.

[5] Ibid.

[6] Ibid. i. 190.

[7] Briquet, before quoted, p. 218. Bricheteau, Bull. Gén. Thérap. lxi. 417. Debout, Bull. Thérap. Aug. 30, 1863.

[8] Clarus, p. 840.

In the nervous disorder of the digestion which has been described as associated with diarrhœa, opium before meals, or administered in a clyster at bed-time, often affords relief; but I have known cases in which the health was only finally re-established after the use of nux vomica and hydrochloric acid. Patients suffering in this manner should avoid hot fluids at meals, and abstain from tea in the evening. In fact, in most of the nervous affections which have been here described, the use of tea and coffee, and especially of the latter, is injurious, and often serves to excite pain even after it has been allayed by treatment.

The majority of cases of spasmodic pain supervening in the course of gouty attacks are to be treated with large draughts of warm water, and with ether, musk, or camphor. The sense of distension and load at the stomach, and the relief often experienced from vomiting, are not however, to be taken as indications for the administration of stimulant emetics; for in such cases inflammation of the stomach, if not already present in some degree, is very liable to be excited by any undue irritation of the mucous membrane. It is desirable in all cases to promote by hot pediluvia or by mustard cataplasms the return of the disorder to the feet, and cases are recorded where this has been followed by immediate relief to the stomach symptoms.[1]

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[1] See Dr. Copland's Med. Dict. vol. ii. p. 39; also Dr. Garrod, before quoted.

IV.—Gastritis.—Acute Gastric Catarrh.

Synonyms.—Catarrhe de l'Estomac (Pinel); Gastritis Erythematica; Erysipelas Stomachi; Ventriculi Inflammatio (Boerhaave); Febris Stomachi Inflammatoria (Hoffmann); Febris Mucosa (Typhoid?); Inflammatory Dyspepsia (Todd); Gastrite, Embarras Gastrique—French; Magenentzündung, Magen-Catarrh—German.

Varieties.—Gastritis Erythematica, G. Phlegmonodea: *Cullen.* Erythematous Gastritis, Gastritis with Alteration of Tissue, Follicular Gastritis, Gastritis with Alteration of Secretion: *Billard.* Gastrite Primitive, Gastrite Secondaire: *Valleix.* Phlegmonous, Catarrhal, Rheumatic Gastritis: *Hildenbrand.* Catarrhal, Croupous, and Diphtheritic; Inflammation of Lenticular Glands; Inflammation of Submucous Tissue: *Rokitanski.*

Definition.—An acute disorder of the stomach, characterised by depression and prostration, with or without pyrexia, by anorexia, nausea, vomiting, and in severe cases by pain after food, and depending on an inflammatory condition of the mucous membrane.

The terminology and real nature of this affection have been involved in much obscurity from the variety of the affections confounded under this title, including the specific fevers on the one hand, and on the other post-mortem softenings, ulcer, and cancer of the stomach.

There is no question but that acute typical gastritis, unless when caused by acrid poisons, is a comparatively rare affection, and equally so is the disease corresponding to Cullen's G. phlegmonodea, when suppurative action takes place in the submucous tissue. Its milder forms, corresponding to the catarrhal affections of other mucous membranes, are, however, exceedingly common, and constitute the cause of the majority of the acute attacks of indigestion which occur either spontaneously or in the course of other diseases. The distinction, however, between the acute and chronic stages of inflammatory affections of the stomach is not always capable of being drawn with great accuracy, since, as remarked by Chomel, many persons liable to the disease may often suffer from a succession of subacute attacks, which, being excited by slight causes, may imperceptibly pass into one another, and thus acquire the character of a continuous disorder.

Etiology.—This disease is common at all ages, and in both sexes. It is said to have been observed in the fœtus.[1] In infancy[2] also, and at the periods of dentition, it is easily excited by food unsuited to the digestive powers of the stomach, or improperly prepared by suitable mastication; and in advanced life the latter cause, together with the enfeebled nutrition of the organ, render it liable to be affected by similar influences.[1]

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[1] Andral, Path. Interne, i. 17. Rayer, Dict. de Méd. x. 134.

[2] It is important that the junior practitioner should remember, what is seldom mentioned in systematic treatises, that certain causes may affect the milk given to infants,

Weakened states of the general system or of the stomach, which diminish the secretion of the gastric juice, are also frequent causes of inflammatory irritation, owing to the food introduced not undergoing its normal changes, and thus acting as a source of inflammatory irritation. Hence, among its predisposing causes must be enumerated many of those giving rise to general atony, and especially the presence of chronic exhausting diseases, or the period of convalescence from acute disorders.

A similar influence has been attributed to the effects of inanition or starvation, owing to observations of Hunter[2] and Blundell,[3] where the stomach in such conditions has been found softened after death. Andral[4] also met with ulcerations, and other writers[5] have mentioned that nausea and vomiting have been observed under these circumstances. Such a result is, however, by no means constant;[6] and though it is possible that inflammation may in some cases be caused by the participation of the stomach in the mal-nutrition which occurs in all the tissues under such circumstances, as well as by the arrest of its normal physiological functions, yet it is more probable that in most cases it is due to the effect of food imperfectly digested after periods of prolonged inanition.[7]

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through which it often proves a source of severe gastro-enteric catarrh. Menstruation occurring during lactation is one of these; and in infants brought up by hand on cow's milk, vomiting and purging are frequently excited when the animals are fed on turnips or mangold.

[1] These remarks apply to the acuter forms alone. The only two authors, Dr. H. Jones (Dis. of Stomach, p. 74) and Willigk (Prag. Viertel-Jahresch. li. p. 28), who have made observations on this head, have included both recent and chronic catarrh in their statistics; and in these taken collectively, the tendency to increased frequency of occurrence with advancing age is very striking. Willigk's observations, however, only begin with ages from thirty upwards; and of twenty-three cases, by Dr. H. Jones, "eleven were past fifty years, and fifteen past forty years of life." I believe, from my own observations, that the increased frequency of gastric catarrh in the later periods of life will be found rather to affect the chronic than the acuter forms.

[2] Phil. Trans. 1772, "Observations on certain Parts of the Animal Economy."

[3] Quoted by Dr. Hodgkin, "Morbid Anatomy of the Mucous and Serous Membranes," ii. 309.

[4] Path. Interne, i. 15. Essai d'Hématologie Pathologique, p. 82.

[5] See especially a letter from Mr. Malcolmson to Lord Hardinge, on the effects of a diet of bread and water on prisoners in causing total loss of appetite, constipation, or diarrhœa with slimy discharges, together with fever, a swollen red tongue, and great prostration. Quoted by Budd, loc. cit. p. 96, who gives other cases.

[6] See Chossat, "Rech. Exp. sur l'Inanition;" Barras, loc. cit. p. 522; also Taylor's "Medical Jurisprudence."

[7] Chossat found that pigeons in a state of starvation could not digest the food given them, and that under these circumstances they suffered from diarrhœa; and other illustrations of the same fact occur in the histories of persons who have suffered from starvation. Barras (ii. 168) says that after the season of Lent many persons suffer from indigestion. Two facts, observed by Bidder and Schmidt, deserve to be borne in mind in relation to this question, viz. that after a moderate period of fasting the secretion of the gastric juice is increased, but that after longer abstinence it is diminished.

As exciting causes must be mentioned irritants of all kinds, including those whose action is purely mechanical, but especially the mineral and vegetable acrid poisons, and particularly arsenic,[1] and tartar emetic,[2] mustard, and ipecacuanha when administered as emetics.

In the same category are to be placed substances of an unsuitable character taken as food, such as decomposing meat or vegetables, or shell-fish in some special conditions—which latter seem to have a peculiar efficacy in this direction. The same effect may, however, be due to alimentary substances which are not directly injurious, taken in excess of the digestive powers of the gastric juice, though their influence in this respect must to some degree depend upon their relative digestibility. Similar consequences may ensue from causes operating through the nervous system suddenly arresting the process of digestion, and thus reducing the food which has been taken to the position of a foreign body, and consequently an irritant to the stomach.

Drinking largely of cold water when the body has been heated is also mentioned as a cause of catarrh of the stomach. Sudden changes of temperature have had a similar influence attributed to them;[3] and climatic conditions appear sometimes to act in the same direction, for catarrhal affections of the stomach are most common in changeable weather, with cold and high winds, as in the spring and later autumn,[4] and also during the severe heats of summer and early autumn.

There appears to be a certain amount of evidence to show that epidemic influences have some share in producing this disorder. Thus Sydenham describes in the years 1669-70-71-72, as coincident with dysentery, and following an epidemic of "cholera," a fever setting in with gripes, headache, a moist tongue with a thick fur and aphthæ, *cured in six days by purging and low diet.*[5] Barras[6] remarked, that during the cholera epidemic of 1832 affections of the stomach were very common. P. Frank[7] ascribed them to a *constitutio annua*, and

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[1] The use of arsenical paper-hangings has also caused the same condition, an instance of which has come under my knowledge, where a previously healthy child was seized with violent vomiting, in which blood was brought up, while sleeping in a room so papered. Dr. King Chambers has narrated a similar case—"Indigestions," p. 217.

[2] Andral, Clin. Méd. i. 246.

[3] Guipon (Traité de la Dyspepsie, Obs. 21, p. 329) has recorded a case of a workman who, after exposure to the heat of a furnace, was seized with acute vomiting and pain at the stomach, and this accident was repeated several times.

[4] Broussais (Hist. des Phleg. ii. 456, ed. 1822) says that inflammatory affections of the gastro-intestinal mucous membrane were very common in the Venetian Friuli, but many of his cases can hardly be considered as examples of simple gastritis, and would now be considered as pneumonia, typhoid fever, or acute tuberculosis. Brighton has universally the reputation of making many "bilious" during early periods of their residence there; and Dr. J. Todd (Cyc. Pract. Med. art. "Indigestion") says that these disorders are common in Turkey, Greece, Italy, Spain, Nice, Genoa, and Marseilles; and that they often follow the *bise* in Switzerland, the *mistral* in Provence, and the *tramontana* in Italy. Willigk (Prager Viertel-Jahreschrift, li. p. 28) gives for 327 cases of acute and chronic catarrh combined, observed in five years, the following relative proportions between the number of cases occurring in the different seasons:—Spring, 6·2; summer, 3·4; autumn, 2·9; winter, 2·5.

[5] Syd. Soc. Trans. i. 177, 181.

[6] Loc. cit. ii. 161, 162.

[7] De Curandis Hominum Morbis, i. 73—75.

Schmidtmann[1] to a *constitutio gastrica*, independent of any special kind of weather. Chomel has remarked the coincidence with cholera of the cases of vomiting which he terms "*dyspepsie acide grave.*" During the height of the epidemic in August 1866, I was much struck with the frequency of subacute inflammatory affections, corresponding in their symptoms to the state known by the French as "embarras gastrique;"[2] and Barthez and Rilliet[3] consider the probability of this affection and of other forms of gastro-intestinal catarrh taking place under epidemic influences as very strong. It has also been stated that there is a special proclivity to gastro-intestinal catarrh during some epidemics of typhoid fever.[4]

There are several other diseases with which inflammatory conditions of the stomach so frequently concur, that they may almost be considered as part of the general disorder. Many belong to the acute febrile affections; and although the impairment of the functions of the stomach in them is not always due to changes of an inflammatory character—nor is it easy to explain why these should be prominent in some cases and absent in others—yet the influence of some is so well marked, that they deserve to be mentioned as almost constant exciting causes of gastric disorder. Among these may be mentioned cholera[5] and scarlatina,[6] erysipelas[7] and measles;[8] and I have observed the same conditions in diphtheria,[9] variola,[10] puerperal fever,[11] phlebitis, pneumonia[12] and, pyæmia, and, with a less frequency, in typhoid fever. It is also a common complication of pulmonary tuberculosis, this condition having been met with in 28 per cent. of a series of cases of acute and chronic catarrh tabulated.[13]

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[1] Summa Obs. Med. iii. 300. There is no doubt that some of the epidemics spoken of by older writers, as by Elsaesser, Sarcom, Roederer, and Wagler ("De Morbo Mucoso"), P. Frank and Reil, included cases of typhoid fever, "or of typhus complicated with dysentery" (Murchison). See J. Frank, Prax. Med. Univ. Præcept. 1811, vol. i. pars i. p. 244; also an analysis of this subject by Dr. Murchison, "Treatise on Continued Fevers," p. 393.

[2] See also the report in the Medical Times and Gazette, July 1866, of the frequency of "embarras gastrique" during the outbreak of cholera at Amiens. A similar frequency of this complaint has reappeared during the present autumn (1871), at least in my private practice. I have not seen signs of gastric catarrh so common since 1866. Cholera has been prevalent in Europe, though not in England.

[3] Traité des Malad. des Enfants, i. 717, 732, 739, &c.

[4] Schmidt's Jahrbücher, 1863, pp. 123, 243. Bericht über die Krankenhäuser Wieden.

[5] Several observations on the effects of cholera in causing acute gastritis are also to be found in Andral, Clin. Méd. ii.

[6] Brinton, Diseases of Stomach, p. 57.

[7] Bamberger, P. Frank, Fenwick, Med.-Chir. Trans. xlvii.

[8] Barthez and Rilliet (Malad. des Enfants, iii. 271).

[9] Sir W. Jenner has also seen diphtheria of the fauces associated with false membranes in the stomach ("Diphtheria," 1861, p. 4). This does not appear to be common, and, from Sir W. Jenner's observations, it is probable that its occurrence is somewhat influenced by epidemic character. (See also Squire, art. "Diphtheria," vol. i. p. 401.)

[10] See also Andral, Prec. Path. Anat. ii. 226.

[11] In a series of observations where my attention was specially directed to this point, I observed catarrhal affections of the stomach in only four out of nine cases of this disease.

[12] Originally observed by Dr. Stokes, Cyc. Pract. Med. iii. art. "Gastritis."

[13] See chapter on Chronic Catarrh.

Inflammation of the stomach has also been observed to follow the retrocession of gout and of acute rheumatism.[1]

It may also be considered a question deserving further elucidation whether some of the cases of vomiting in pregnancy, hitherto set down to reflex irritation, may not depend on alterations of a similar kind.[2]

The liability of the stomach to suffer from other causes inducing constitutional irritation and febrile action is illustrated by Abernethy's observations,[3] who was well acquainted with this effect of general disturbance of the system.

The question of the possibility of nervous disturbance chiefly arising from moral emotions, acting as exciting causes of the disease, has been already alluded to. There are, however, as has been before remarked, but few authentic or uncomplicated instances of this nature recorded. That they may act as indirect causes by arresting digestion, is very probable; but further proof appears to be required before their direct influence can be regarded as fully established.[4]

THE SYMPTOMS of recent or acute catarrhal affections vary considerably in intensity according to the degree of severity of the attack. They may be generally comprehended under the following category :—Uneasiness, distress or pain at the epigastrium—the latter symptom being however occasionally wanting, or not present to any marked degree, even in some of the severer forms of the disease; anorexia more or less complete, vomiting, thirst, general malaise or prostration, headache, febrile reaction of variable intensity, thirst; constipation in some cases, diarrhœa in others. Beaumont's observations have shown that in slighter cases of this nature local uneasiness may be completely absent, and the disorder of the stomach may only be revealed by general malaise accompanied with slight headache. There are also differences observable in the character of the attacks, and the disorder may be described as existing in certain typical forms, between which, however, every shade of variety or resemblance may in different cases be found to exist. The principal of these are:

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[1] The former has not been proved by post-mortem evidence. Of the latter a fatal case is recorded by Andral, Clin. Méd. ii. p. 11. I have observed a case where the retrocession of the pains from the joints was followed by vomiting and diarrhœa, and a similar one is recorded by Chomel, Des Dyspepsies, p. 137. I have also observed in three cases in private practice intense gastric catarrh precede, in two cases by many days, and in one for many weeks, an attack of acute rheumatism.

[2] The majority of cases where examinations after death from this cause have been recorded speak of the stomach as showing little or no signs of disease. Virchow, however (Ges. Abhand. 778), has shown that in pregnancy the liver sometimes shows the same alterations as have been commonly noticed in the kidney, and which, under the title "Cloudy Swelling," are recognised as indicative of an inflammatory condition. This state will be described in the account to be given of the pathological changes found in catarrhal inflammation of the stomach, as frequently forming the most characteristic appearance present. In the Dict. des Sciences Méd. vol. xvii. p. 382, art. "Gastrite," is recorded, by Guersant, a case of a woman dying from vomiting in pregnancy, in whom the stomach was found "*très-blanche*, un peu *plus épaissie* que dans l'état naturel, et *recouverte d'une mucosité abondante.*"

[3] Rayer (Dict. de Méd. x. 136) says that he has observed that inflammations of the joints, the kidneys, bladder, and serous membranes had an important influence in determining gastro-enteric inflammation, which was acute or chronic according to the severity and duration of its cause.

[4] With the exception of the case previously quoted from Andral, the recorded observations of this nature are chiefly in older writers. (Hoffmann, De Inflamm. Ventris Frequentiss. Op. vol. vi. 223–227; Blasius, Obs. Med. Anat. Rariores; Barry, Acta Reg. Soc. Med. Hannov. vol. iii., all cited in Copland's Dictionary.)

(*a*) Acute indigestion and the "embarras gastrique" of the French authors.[1]

(*b*) Febrile forms in which the fever is secondary to the disorder of the stomach.

(*c*) Acute catarrh in infants.

(*d*) Severe inflammation resulting from irritant poisons.

(*e*) Catarrhal affections of the stomach, complicating the exanthemata and other acute diseases.

(*f*) Acute catarrh of the stomach arising from alcoholic excess.

(*g*) Gouty inflammatory affections of the stomach.

(*a*) Acute indigestion may assume various degrees of severity according to its cause or the previous health of the patient. In some cases it may present only the phenomena of a trifling "bilious" attack; in others it may last many days or weeks.

Its origin will ordinarily be found in some of the causes temporarily disturbing the digestion; a moral emotion, or severe exercise after a meal, indigestible food taken in excessive quantity, or food against which an idiosyncrasy exists on the part of the patient, are, however, among its most frequent causes. The first symptoms generally are a sense of fatigue, together with malaise, aching in the back or limbs, and depression of spirits; these are soon followed by epigastric uneasiness and distension, and sometimes by severe cramp-like pain in the stomach. During these attacks there is often a sense of faintness, the extremities are cold, and the pulse is weak, fluttering, and depressed, and the patient is often bathed in cold perspiration. Headache soon supervenes, generally frontal in position, sometimes of considerable severity, and not unfrequently associated with intolerance of light and sound. Nausea with increased flow of saliva follows, and the offending meal is rejected, accompanied by a great quantity of acid fluid; and with its expulsion the symptoms may cease. In other cases, instead of being vomited, the irritating matters pass into the intestines. Griping and colicky pains then ensue; in some cases a spontaneous diarrhœa is set up which carries off the peccant material, but in others constipation, associated with flatulence and spasmodic contractions of the intestines, continue until the bowel is evacuated by a purgative. In the latter cases, pain, sometimes acute, is felt at the epigastrium; or there may be only an excessive sense of uneasiness and of weight or load at the præcordial region. There are complete anorexia and loathing of food, and nausea continues, often attended with ineffectual attempts to vomit. The tongue becomes loaded with a thick creamy fur; and though the amount of this varies in different cases, being sometimes thin enough to allow the enlarged papillæ to appear through, it always retains its soft, moist, milky appearance; the breath is offensive, and thirst is generally a marked symptom. In other and severer cases the circulatory and nervous systems may participate in the general disorder; palpitation, dyspnœa, faintness, vertigo, or a confusion of ideas may supervene; and when in the case of elderly people an excessive amount of flatus is generated, cerebral congestion may occur to an extent sufficient to simulate an apoplectiform attack; while in children, and sometimes also in females, the implication of the nervous system may induce convulsive affections of the epileptiform character.

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[1] Many of these authors consider "embarras gastrique" as a distinct disorder, having nothing in common with the inflammatory processes. I cannot but regard this question as set at rest by Beaumont's observations, and think that the difference between these and severer affections is only one of degree—an opinion which is confirmed not only by their etiology, but also by the effects of treatment.

The headache which appears in the course of the slighter attacks of this nature often assumes a form with somewhat characteristic features, and which is familiarly known as the "*sick headache.*" It is most common when acute exacerbations are superadded to the ordinary forms of atonic dyspepsia; and hence it is most liable to affect those who are out of health, and whose digestions are weakened by sedentary employment, and who have a tendency to costiveness. It occurs, however, also in persons of apparently vigorous health, sometimes without apparent cause, but most usually after some indiscretion in diet, or after some of the causes liable to arrest the digestive process.[1]

The most usual time of its appearance is some hours after food has been taken, and very commonly the patient wakes with the pain at an early hour in the morning, especially when the last meal has been a late and indigestible supper. It may, however, supervene at any hour of the day. The attack is usually preceded for a longer or shorter period by some indistinctness of vision, sometimes affecting half the field of vision of one or both eyes; at other times diplopia occurs, or sight is disturbed by muscæ or by dazzling spots of light. Vertigo and noises in the ears may also appear among the prodromata. These are usually soon followed by pain in the head, at first slight, but rapidly increasing, until it becomes of great severity, which most commonly affects one or both temples, the frontal or in rarer cases the occipital region. There is often acute throbbing pain in the eyeballs, which are tender to pressure; though when the pain in the head supervenes, the indistinctness of vision usually disappears. If the pain lasts long the scalp sometimes becomes tender, and it not unfrequently remains so for some time after the attack. During the paroxysm the surface of the head, and particularly the forehead, is often cold, and in some cases the pain itself may be partially relieved by hot fomentations; at a later period the skin of the head generally becomes hot.

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[1] Fothergill, who first described this headache (Med. Obs. and Enq. vol. vi.), attributed to "butter, fat meats, spices," and "meat pies," a special faculty in its production. Wood (Practice of Med. i. 564) says that it is frequently caused by excesses in the use of tea and coffee, but especially of the latter.

Other symptoms accompany the attack. The physical and moral depression is, in severe cases, extreme; light and sound are equally intolerable; sighing, yawning, or shuddering are often present, together with an extreme sense of general chilliness, amounting at times to rigor. Nausea is very common, and vomiting of acid food sometimes occurs—this appears sometimes, but by no means constantly, to relieve the patient. In other cases there is a great sense of uneasiness in the lower bowels, leading to ineffectual attempts at evacuation, and the only effectual mitigation in such cases is that produced by a purgative. The attack may last only for an hour or two, or may persist for twenty-four or forty-eight hours. When it is severe, complete relief is rarely obtained until after sleep has been procured, though this is unattainable during the height of the paroxysm. After sleep the patient awakes either free from pain, but feeling weak and nervous, or sometimes with a dull aching in the head, which gradually disappears. A loss of appetite, and diminished digestive power, which sometimes entail a liability to a speedy recurrence of the attack, often remain during some days.

The nature and immediate causes of these attacks have been a subject of much discussion. Dr. Anstie[1] has recently adduced some good reasons for regarding the pain, when seated in the anterior part of the head, as a neuralgic affection of the fifth nerve, and it is not impossible that this may be its real explanation, since other neuralgiæ of this nerve have been observed to follow disturbances of the stomach. It would appear that these headaches may be immediately produced by undigested food, either remaining in the stomach or which has already passed into the intestines; and this opinion is corroborated by the methods through which relief is usually obtained. Whether any special conditions of the food or of the secretions are concerned in their production must remain, as heretofore, in the absence of positive data, a matter of speculation.

Anorexia, pain, and thirst, with a loaded tongue and great general depression, may sometimes continue for days, owing to undigested food being retained in the stomach, and may disappear after this has been evacuated by an emetic, but in other cases the irritation remains long after its cause has been removed. The persistence of the symptoms above indicated, with certain others superadded, then forms the "*status gastricus*," "*saburral condition*," or "*embarras gastrique*" of the German and French authors, which may in most cases be traceable to some of the causes above indicated, but in others it occurs apparently spontaneously, or it may result from fatigue, over-anxiety, or probably from some of the epidemic influences before alluded to. There is then tenderness, load, and uneasiness at the epigastrium, together with great disgust for food, which, when taken, increases the distress, or causes nausea, and sometimes vomiting of mucus, or bile, or food acid from fermentation, together with acid watery fluids. Thirst is a marked symptom, and there is sometimes a craving for acid drinks; the tongue is more or less thickly covered with a moist white or brown fur; there is a bitter, nauseous, and sometimes metallic taste in the mouth, and an increased flow of saliva has been occasionally noticed. Fetid and acrid eructations and heartburn are often complained of, and the breath is heavy and offensive. The bowels are, as a rule, confined. In some cases diarrhœa may have been present at the outset, with colic and griping, but on ceasing it is followed by constipation; in rarer instances, in which catarrh of the intestinal canal is also present, it persists throughout, and is then attended with griping and with pale watery stools, which often irritate the anus and rectum when passed. There is great physical and intellectual oppression, together with a sense of fatigue and weakness, which may be the sole symptoms felt by the patient, or for which relief is sought; or these may be accompanied by a dull, confused headache, becoming sharper at intervals and not relieved by sleep. Sleep is unrefreshing, and is disturbed by dreams or nightmare. There are often rigors and slight horripilation of the skin, especially towards evening, with a certain amount of febrile reaction, ending sometimes in acid perspiration during sleep: an icteric tint of the conjunctivæ is very common. The pulse, except during febrile accessions, is generally depressed and weak, and slower than natural, though easily accelerated on slight exertion. If fever supervenes, it becomes quick and full, but easily compressible. Urticaria and herpes sometimes complicate these attacks: the former is often caused by shell-fish, or by substances against which the patient has a special idiosyncrasy;[1] the latter, when appearing in connexion with acute attacks of indigestion, mostly affects the alæ nasi, the lips, and chin, and more frequently results from the use of malt liquors in persons with whom these habitually disagree, than from any other single cause with which I am acquainted.

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[1] "On certain Painful Affections of the Fifth Nerve," Lancet, 1866, ii. 32.

The urine is usually scanty, acid, high-coloured, and loaded with lithates on standing; it may in some forms of acute indigestion occasionally present traces of albumen.

The duration of such an attack is uncertain: when appropriately treated, it usually terminates in a few days, though a certain irritability and weakness of digestion may continue for some time after; but when neglected, or when food is indulged in as usual, or if alcoholic stimulants are taken in excess to relieve the flatulence or feelings of prostration accompanying the attack, it may be prolonged almost indefinitely in a subacute form.

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[1] Mushrooms, cucumbers, almonds, oatmeal, pork pie, and mackerel (Budd, loc. cit. 266). Cubebs also has been known to cause it (Wood, Pharmacologia, i. 331). In some of these cases the invasion of the nettle-rash is not always accompanied with the signs of gastric disorder here mentioned. Herpes zoster, though preceded often by severe constitutional disturbance, is not so common a phenomenon of these attacks.

(*b*) There are, however, severe forms of the disorder, marked by considerable febrile reaction, which are very difficult to distinguish from febricula, or sometimes from early stages of typhoid, but in which the febrile reaction appears to be in reality attributable to the stomach. One class of these cases is marked by epigastric pain of some severity, which is generally central, but which sometimes radiates into the hypochondria and extends to the back. The sensation is sometimes one of heat or burning, at others of load or constriction; but as a rule it does not present the same degrees of intensity as are observed in nervous gastrodynia or in the pain from ulcer and cancer. In other instances, however, pain is not complained of. Vomiting also occasionally occurs, and may be almost constant after everything taken, and be brought on even by the smallest amount of liquid—the matters rejected being mucus, sometimes tinged with blood; or bile in considerable quantities; and retching may continue even after the stomach has been emptied. The tongue in these cases may present the loaded "saburral" state before described, but it tends in a day or two to become red, raw-looking, and sometimes fissured; the papillæ are large and red, and the lips dry and cracked. Sordes sometimes appear on the teeth.

Thirst is usually considerable, and the appetite is completely lost.

Constipation generally persists, and sometimes with considerable obstinacy; but diarrhœa may occur, though this is comparatively unfrequent.

Rigors, to a mild degree, usually continue throughout the whole course of this affection. The skin is often hot to the hand, but except in cases of children the elevation of the temperature is rarely considerable, and seldom above 100° Fahr.; but in children it may reach 103° or 104° Fahr. There is generally a considerable exacerbation of fever in the evening, and the diurnal remissions may be almost complete. The pulse is frequent, but weak and compressible.

Prostration with restlessness, and pains, though only of moderate severity, in the back and limbs, also continue throughout the attack; and the headache, which is ordinarily frontal, is frequently severe. Sleep is usually disturbed; and in children delirium may supervene, or a semi-comatose condition may be observed, and strabismus occasionally occurs. I have never observed either sluggishness, or marked contraction, or inequality of the pupils. The urine is scanty and high-coloured, and deposits lithates.

Cough is spoken of as a common complication. I have not observed this unless when from a common exciting cause, such as cold, a bronchitis has been set up simultaneously with the gastric catarrh. Cough, associated with pyrexia, should always be regarded as a symptom requiring a careful and suspicious investigation of the lungs, for the condition which has now been described is often the accompaniment of early stages of phthisis.

The duration of this complaint, in its acute form and under proper treatment, is seldom longer than a week or ten days; but if treated at the outset with tonics and alcoholic stimulants, it is liable to become almost indefinitely protracted, and to pass into some of the more obstinate forms of chronic catarrhal inflammation.

(*c*) In young children, especially in infants under six months, or at the period of weaning, improper or excessive food sometimes causes a general catarrh of the whole gastro-intestinal canal. This may find its chief expression in diarrhœa, which frequently precedes the vomiting; but in many cases the latter is often an important, and even a dangerous symptom. There may be but little fever, and even when the skin over the abdomen is hotter than natural, the extremities and lips may be cold and bluish; and though the abdomen is sometimes tender, this is not constantly observed. Pain is, however, frequently evinced by cries, especially before the evacuations. These are liquid, watery, offensive, acid, and often grass-green in colour; they generally contain masses of coagulated casein, and are often attended with straining or tenesmus: but when the attack is severe and the child much prostrated, they may be passed apparently unconsciously. The vomited matters consist of the coagulated milk, returned in an intensely acid condition, and accompanied with much acid watery fluid. Thirst is frequently excessive, but fluids taken are often rejected almost as soon as swallowed. The patient rapidly loses flesh, and great prostration sets in early, so that the infant may have difficulty in sucking, though it drinks with avidity. The pulse becomes weak and fluttering, the fontanelles are depressed, the countenance is pale, the eyes are sunken, and the features have a peculiar pinched, sharpened appearance. Somnolence, passing into coma or convulsions, may at times complicate the other symptoms, but the latter phenomena are not frequent. The cause of this form is acute, and if not early checked, it tends towards a fatal issue.

I have occasionally observed affections of this nature, though of somewhat less severity, and occurring at the period of the first dentition, alternate in a remarkable manner with eczematous affections of the skin.[1]

It may be remarked that the symptoms of the choleriform diarrhœa of children correspond to a great degree, as far as regards the stomach, with those observed in true cholera in the adult. In the majority of the cases of this disease which have come under my observation, the stomach participated markedly in the catarrhal condition of the intestines,[2] and the thickly-furred tongue in many corresponded most closely with that of the "saburral" conditions which have been before described.

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[1] In one case, in a strong and otherwise healthy child, the eruption of each tooth was either attended with an attack of acute eczema, or by an attack of vomiting with diarrhœa; and, during one of the latter, the gastro-intestinal symptoms suddenly ceased on the supervention of the eczematous rash. It appears difficult to explain these phenomena, except on the theory of some *materies morbi* in the blood finding an excretory outlet by the skin or mucous membranes.

[2] See report by author on appearances in cholera, Path. Soc. Trans. 1866-7.

It appears, however, unnecessary to enter into fuller details of the symptoms of this affection, though it is not unimportant to bear in mind its pathological relationship to the class of diseases which we are now considering, and to remember that the diseased conditions excited by cholera may persist in the stomach long after the other leading features of the disorder have subsided. This has been observed by numerous writers, especially by Andral, Budd, and Chomel; and their experience can, I have very little doubt from my own observations, be confirmed by any who have had opportunities of following the history of patients who have been subjects of the latter disease. Chomel's[1] "dyspepsie acide grave," with its acid vomitings, is the counterpart of that observed in children; but I have great doubts whether such acidity is the result of hypersecretion, and not rather the consequence of rapid catalytic changes in the food taking place under the influence of the unhealthy secretions of the stomach, especially as in some cases recorded by other observers it was found to be greatly increased by farinaceous food.[2]

(*d*) The symptoms of the typical acute form of gastritis are merely exaggerations of the milder varieties just described, the gradations observed consisting principally in differences in the degrees of severity; but the more marked characters of the disease are rarely met with except when the more violent irritants have been swallowed.

There is usually acute epigastric pain, though some remarkable exceptions have been observed in this respect by Dr. Habershon, even in cases where corrosive poisons have been taken.[3] Its characters are burning and lancinating; it often extends into the back, and, when the affection is severe, it is not relieved by vomiting, and is increased by pressure. It is often accompanied by spasm and rigidity of the abdominal muscles, and is aggravated by each descent of the diaphragm, so that the respiration frequently becomes wholly thoracic.

Vomiting is a constant symptom: it is frequent, and is brought on by the smallest quantity even of cold fluids. There is also violent retching, which continues when the stomach is empty. The matters brought up are mucus, often tinged with blood, or blood blackened by the fluids of the stomach, together with bile and watery fluids. Diarrhœa is present in some cases, together with colic, tenesmus, and bloody stools, especially after arsenic, antimony, or corrosive sublimate have been taken;—absent, or not so commonly met with, after the mineral acids, and caustic alkalies. There is complete anorexia, but great thirst. Prostration is marked, but often combined with agitation and restlessness. The face is pale and sunken, the voice weak or extinguished.[4] The skin is cold, and often covered with clammy perspiration, and the pulse is frequent and small. Hiccup is sometimes a very painful symptom, and may continue after the vomiting has ceased.

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[1] Loc. cit. p. 144.

[2] See Guipon, Obs. 91, p. 436.

[3] Oxalic acid, sulphuric acid, arsenic, and chloride of zinc. (Med. Times and Gazette, Nov. 20, 1859; and Diseases of Stomach, 1866, p. 41.)

[4] It may be in some cases affected by the action of the irritant upon the epiglottis.

The duration of these symptoms is variable. Death may ensue with great rapidity by complete collapse; or the patient may linger for days and die from exhaustion; or long-continued irritability may persist for weeks, and subsequent dangers may ensue from hæmorrhage, or from the contraction of cicatrices resulting from ulcerations in the pyloric region.

(*e*) The symptoms of inflammatory disorder of the stomach complicating other acute diseases are often considerably modified, or even masked, by the course of the disorders in which they occur. Anorexia and thirst are common to those in which we have no evidence to show that the affection of the stomach is of an inflammatory nature. I am inclined, however, to believe that in the majority of cases where, in addition to these, we find a loaded tongue and nausea after food, and even a slight degree of epigastric tenderness, this condition of the stomach is the cause of these symptoms much more frequently than is generally supposed to be the case, and I have had repeated opportunities of verifying this opinion by post-mortem examinations. In some, however—and this is particularly true of variola and scarlatina, and not unfrequently also of pneumonia—other and more distinct symptoms appear, in the form of vomiting. Spontaneous pain is not, however, usually present. Though these evidences of gastric inflammation are often more distinct in the disorders last mentioned than in some other of the acute diseases, yet, with regard to the whole class, the liability to this complication should be always recollected in considering the measures of treatment to be adopted.

(*f*) In the inflammation of the stomach that follows from drink the symptoms are often very obscure. Vomiting, except in the morning, is comparatively rare, and signs of tenderness can with difficulty be elicited on pressure. The loaded tongue, the absolute anorexia, and the thirst, serve however as signs of an inflammatory condition of the stomach, of which confirmation is afforded, in some cases of fatal delirium tremens, by post-mortem evidence, and in others, whose termination is more favourable, by the successful results of treatment directed towards this complication.

(*g*) Gouty attacks of inflammation of the stomach usually occur under two forms. The outbreak of the disorder is frequently complicated with all the symptoms before described as those constituting an acute attack of indigestion, which are sometimes relieved, but at others persist, when one or more joints have become the seat of the characteristic inflammation. The more severe and dangerous forms, however, are those attended by a sudden disappearance of the inflammation of the joints and by a simultaneous accession of epigastric pain and tenderness, together with vomiting, and accompanied by severe prostration, and these symptoms may proceed to a dangerous extent unless relieved by a return of the disease to its previous seat. Nor does the predominance of nervous symptoms in some cases at all preclude the possibility of even these being due to the suddenness and severity of the affection of the stomach, though the extreme degrees of flatulence and spasm which accompany them are in themselves almost sufficient to account for these phenomena.[1]

THE PATHOLOGY of the symptoms here described involves the consideration of the *nature* of the condition in which they originate, and of the *anatomical alterations* by which they are accompanied, and upon which they in all probability depend. The evidence that these changes are of an inflammatory kind is in part directly demonstrable, and in part is the result of induction. In the milder forms we have seldom, if ever, an opportunity of experimentally verifying this opinion by post-mortem examination; but the observations of Beaumont, although too often forgotten in actual practice, seem to set this question conclusively at rest.

Having been conducted on a living subject, they possess the advantage of being records not only of changes affecting the glandular tissues, but also of conditions of perverted vascularity, which in the stomach, as in external parts, are only apparent while the circulation is still maintained, and which speedily become indistinct after life has ceased.

The appearances observed by Beaumont cannot be better described than in his own words:—"There are sometimes found, in the internal coat of the stomach, eruptions or deep red pimples, not numerous, but distributed here and there upon the villous membrane, rising above the surface of the mucous coat. These are at first sharp-pointed and red, but frequently become filled with white purulent matter. At other times, irregular circumscribed red patches, varying in size and extent from half an inch to an inch and a half in circumference, are found on the internal coat. These appear to be the effect of congestion of the minute blood-vessels of the stomach. There are also seen at times small aphthous crusts in connexion with these red patches. Abrasion of the lining membrane, like the rolling up of the mucous coat into small shreds or strings, leaving the papillæ bare for an indefinite space, is not an uncommon appearance. These diseased appearances, when very slight, do not always affect essentially the gastric apparatus; when considerable, and particularly when there are corresponding symptoms of disease, as dryness of the mouth, thirst, accelerated pulse, &c., *no gastric juice can be extracted.*"[2] "Complained of headache, lassitude, dull pains in left side and across the breast, tongue furred into a thin yellowish coat and inclined to dryness. Eyes heavy and countenance sallow. The villous membrane of the protruded portions of the stomach very much resembled the appearance of the tongue, with small aphthous patches, in many places quite irritable and tender."[3]

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[1] See Garrod, On Gout, p. 505.

[2] Loc. cit. p. 99.

[3] Loc. cit. p. 171.

"The gastric fluids extracted were mixed with a large proportion of thick ropy mucus, and considerable muco-purulent matter slightly tinged with blood, resembling the discharge from the bowels in some cases of chronic dysentery." . . . "Flavour peculiarly fetid and disagreeable, alkalescent, and insipid."

In other places he mentions the phenomenon of minor degrees of hæmorrhage as not uncommon,—"grumous blood exuding from several small points of the membrane."

It is remarkable that in many instances when these appearances were well marked, the symptoms experienced by the patient were but slight; and hence, *à fortiori*, we may conclude that when the latter are more severe, the anatomical changes are more considerable, though direct evidence of this is often unattainable.

The appearances[1] thus described are, however, much less distinctly seen in post-mortem investigations; and the difficulty also of appreciating such evidences as are derived from the apparent vascularity of the organ is often very considerable.

As regards the phenomena of the latter class, two propositions, the converse of one another, may be laid down as true with certain limitations to be immediately explained. Firstly, that considerable vascularity is not necessarily evidence of inflammatory action; and secondly, that an almost entire absence of this appearance by no means excludes the pre-existence of this process.

Evidence in favour of the first statement has been abundantly accumulated since the time of Morgagni,[2] and more fully by Dr. Yellowly in 1813,[3] who was shortly followed by Billard,[4] by Trousseau and Rigot,[5] and by Andral.[6] These authors have shown that partial hyperæmias and also general straining of the stomach may be determined by the position of the body, by the fluidity of the blood, and also by obstructions to its return from the abdominal organs existing in the vena portæ, the heart, or the lungs. Andral adds, that when death takes place during the act of digestion, hyperæmia of the stomach is generally found; but numerous exceptions will be found to this rule, when the examination has been made some hours after death.

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[1] Pustular appearances of the glands have, however, been described by Rayer as occurring in the stomach (Dict. de Méd. x. 120), and also by Wahl (Virchow's Archiv, xxi. 579). In the latter case mucedines were found in the glands. The appearance is, however, rare, and must not be confounded with enlargement of the solitary glands hereafter to be noticed. A similar appearance is described by Dr. Church, Path. Soc. Trans. xx. 165, when the mucous membrane of the stomach presented an appearance as if covered by small-pox pustules, which seems to have been due to enlargement of the tubular glands, but without degeneration of their epithelium. The glands contained bacterides, probably of post-mortem origin. Purpura had been present, and also vomiting for a month before death.

[2] De Caus. et Sed. Epist. xxix.

[3] Med.-Chir. Trans. iv.: "Observations on the Vascular Appearance in the Human Stomach which is frequently mistaken for Inflammation of that Organ."

[4] De la Membrane Muqueuse Gastro-Intestinale. 1825.

[5] Arch. Gén. xii.

[6] Various places in "Clinique Médicale," and in "Prec. Anat. Path." 1829.

Hyperæmia, of inflammatory origin, is almost invariably purely capilliform and punctiform. The latter appearance is due to small extravasations in the mucous membrane, and may arise from mechanical congestion as well as from inflammatory hyperæmia; and frequently the punctiform redness persists when the general injection, which may be reasonably presumed to have been present, has disappeared. Venous congestion and general imbibition can never, taken alone, be considered as signs of pre-existing inflammatory action. Nor is it always easy to distinguish, apart from other phenomena, the redness of congestion due to impeded return, from that which arises from inflammatory hyperæmia.

It appears, however, equally important to insist on the fact that although during life inflammation of the stomach is probably *invariably* associated with hyperæmia, yet that *post-mortem* pallor of its mucous membrane is no sign of the absence of previous inflammatory action; but that in here, as in other mucous surfaces,[1] even when inflammation has existed, the blood after death leaves the small superficial vessels.

It is only when stasis has existed to an extreme degree, or when punctiform extravasation has taken place from the capillaries, that the signs of inflammatory hyperæmia persist long after death; and even when present they seldom, except in cases of extensive inflammation from irritant poisons,[2] occupy more than patches of the surface.

Other changes, however, exist, which furnish safer criteria for the diagnosis of inflammation than can be derived from the absence or presence of vascularity taken alone. In the slighter forms they are, it must be confessed, somewhat difficult to distinguish, differing, as they do, only by a question of degree from those which occur in the physiological process of digestion, in which not only the vascularity, but also the colour and consistence of the membrane are affected.

These changes consist in an increased opacity, together with swelling and with varying degrees of diminution of consistence of the mucous tissue. The two first of these are distinctly described by Beaumont; and the increased opacity gives to the mucous membrane (apart from the colour produced by hyperæmia) a dead white appearance, corresponding to the "cloudy swelling" of Virchow which is observed in the kidneys in acute Bright's disease.[1]

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[1] *E.g.* in the conjunctiva and in the skin after erysipelas. Andral, Clin. Méd. ii. 177. Congestion of the stomach to an amount sufficient to cause hæmatemesis may leave no traces after death. The bronchi are almost the only exceptions to this rule; but then it should be remembered that extensive bronchitis is almost always attended with congestion of the lungs, which prevents the blood escaping by the pulmonary veins, into which those of the bronchi open. The fading of colour is probably due to the contractile tissue accompanying the capillaries, but in the stomach it is also owing to post-mortem imbibition, and to the effects of the gastric juice. For further confirmation of this opinion the author ventures to refer to a previous work, "Diagnosis and Treatment of Dyspepsia," 2d Ed. pp. 174, 175.

[2] From many of these, as in the cases of arsenic, tartar emetic, and cyanide of potassium taken in large doses, hyperæmia is almost constantly present; but from others, as from phosphorus (Virchow, "Der Zustand der Magen bei Phosphor Vergiftung," Archiv, xxxi. p. 399), it may be absent, and yet other signs of inflammatory action, presently to be noticed, may exist to a marked degree.

Microscopic examination of the mucous membrane in this condition shows that the secreting cells, and also the nuclei, are swollen, irregularly distending the tubules, and are filled with granular matter soluble in liquor potassæ and dilute acids, which gives them, by reflected light, and as seen with a low power, the appearance of white lines, while by transmitted light they appear unnaturally dark and opaque. The cells also often contain fat globules in variable quantity, but in severe cases they frequently break down without undergoing fatty degeneration, and the tubes become more or less filled with granular débris and detritus.

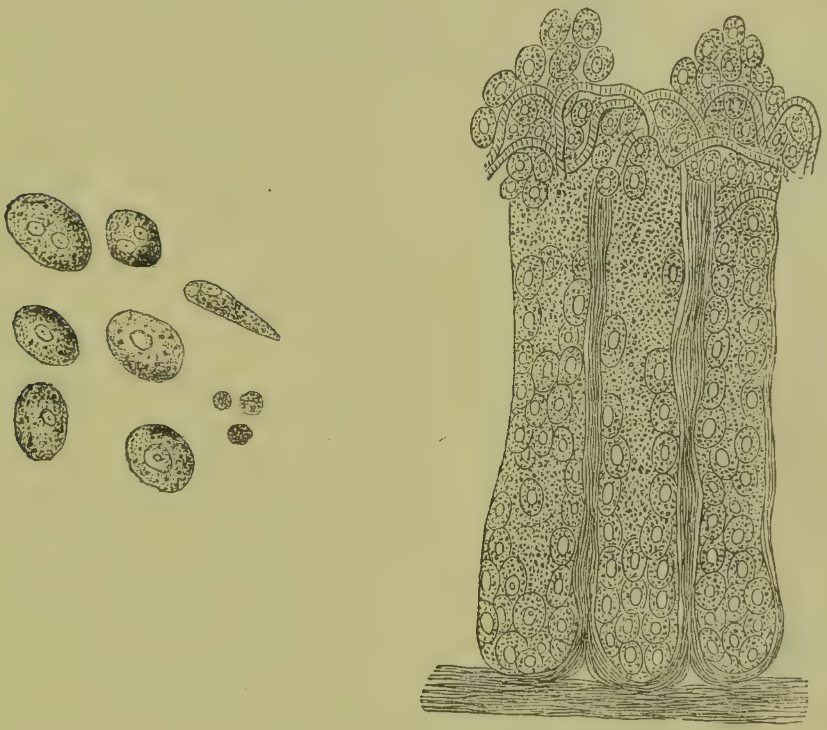

FIG. 2.—Glands and epithelial cells and free nuclei in a case of acute catarrh. The cells and nuclei are swollen and granular. The glands are irregularly distended, and contain a quantity of granular matter free. Enlarged capillaries are seen ramifying at the surface of the membrane.

It is to this distension of the glands, by an abnormal accumulation of protein matters in their interior, that the swelling of the mucous membrane and the pustular appearance observed by Beaumont are chiefly due. The normal secretion of gastric juice is arrested by this state, but at the same time there is produced a considerable amount of tenacious alkaline mucus, containing large quantities of the morphological elements of the interior of the glands, which are generally separate, but sometimes adhere in masses, and then resemble the casts of the tubes excreted in similar conditions from the kidney.[1]

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[1] A case of this kind is recorded by Guersant, art. "Gastrite," Dict. Sciences Méd. xvii. 576, where a young lady died after long-continued vomiting, together with fever and abdominal pain. The mucous membrane was covered with a tenacious mucus, the glands were prominent, and the membrane thickened and moist. The membrane was *whiter than in the natural state, and of the colour of lard.* The vessels on the external surface were gorged with blood. The other organs were healthy. Guersant, not recognising the inflammatory characters of this affection of the stomach, speaks of the disease as one *incertæ sedis.* Carswell, Illust. Elem. Forms of Disease, recognised the occasional *pallor* of inflammatory softening of the stomach.

The softening of the mucous membrane which accompanies these changes is totally distinct from the post-mortem softenings, which are distinguished by the transparency of the tissue. It rarely exists to any marked degree, except in extreme cases, but there is always a certain diminution of resistance to the finger-nail or to the scalpel, which materially assists, when conjoined with opacity and thickening, in distinguishing this condition. Louis' test of the extent to which it can be torn from the submucous tissue is a less available one, and applies rather to states of post-mortem solution than to this condition.

Coincidently with these changes there is a considerable increase in size if not in number of the solitary and lenticular glands, which then appear as small white specks, varying in size from that of a poppy to a millet-seed, thickly scattered over the surface. They are most abundant in the pyloric portion of the stomach and also in the duodenum. A considerable thickening of the interstitial tissue simultaneously occurs, owing to its becoming infiltrated with cell-structures similar to the "lymphatic" elements existing throughout the intestine.[2] They are imbedded in an alveolar network, but are not separated by any distinct line of demarcation from the tubular structures around, which are sometimes widely separated, and more or less obscured by this growth; and these changes greatly increase both the density and the thickening of the mucous membrane.

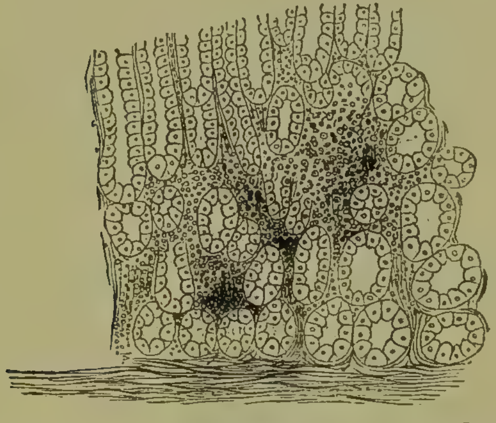

FIG. 3.—Interstitial growth of lymphatic elements between gland-tubes.

In some cases these structures ulcerate, and it is to this cause that the majority of the so-called follicular ulcers appear to be due. These form little cup-shaped depressions scattered more or less thickly over the surface of the mucous membrane, rarely exceeding at the surface a diameter of two or three lines, and seldom extending deeper than the submucous tissue. Their base is found to rest on a tissue infiltrated with lymphatic cells and with the granular débris of these, which may be generally also noticed for some distance in the surrounding tissue.

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[1] Described by Dr. Cayley, Beale's, Archives, i. 198; also by Dr. Fenwick, loc. cit. in scarlatina. I have seen them in the catarrhal affection of the stomach in diphtheria, and also in catarrhal affections of the intestines. They are less frequent either in the stomach or intestines in cholera.

For further descriptions of these appearances see a paper by the author, Med.-Chir. Trans. vol. xli. and Dyspepsia before quoted; also Dr. Schlaepfer, in Virchow's Archiv, vol. viii.; also Dr. Fenwick, Med.-Chir. Trans. xlvii.; also Virchow, in his Archiv, vol. xxxi., and Grohé, Ib. xxxiii.

[2] His. Untersuchungen über den Bau der Peyerschen Drüsen und der Darm Schleimhaut; Frey, Untersuchungen über die Lymphgefässe des Darm-kanals. (Leipzig, 1863.)

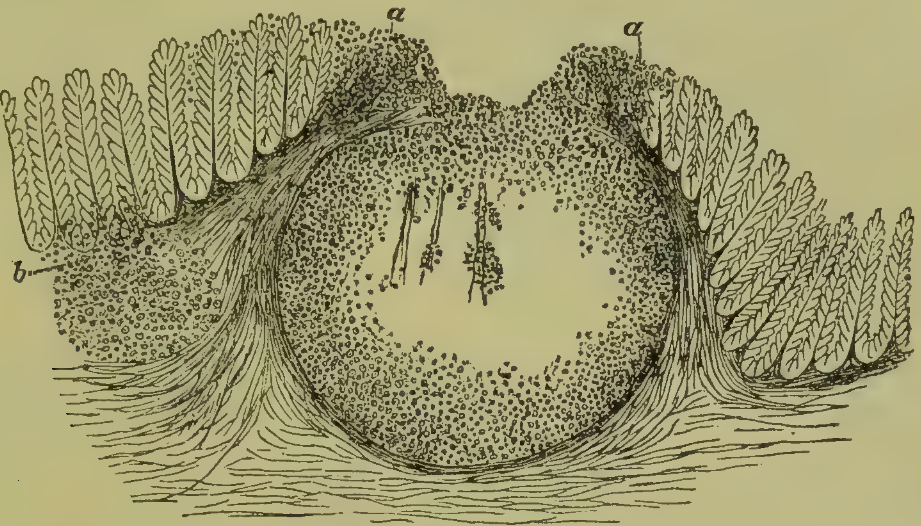

FIG. 4.—"Solitary Gland" reaching surface and commencing ulceration (*a*, *a*). Infiltration of lymphatic elements among gland-tubes. At (*b*) is a mass of lymphatic elements in the submucous tissue beneath the bases of the tubular glands.

Other ulcerations, which are of the nature of erosions, are not uncommon. They are more superficial, but they may attain the diameter of a fourpenny piece. Their edges are often sharply defined, but there is very little thickening around them; I have never seen them extending for any depth into the tissue, and they rarely involve the whole depth of the secreting glands. They appear to arise from a superficial epithelial erosion, sometimes extending rather more deeply into the tissue, and resulting probably from the process described by Beaumont as "stripping and rolling up of the membrane." Early stages of this condition are sometimes found, when in circumscribed patches, giving evidence of acuter inflammatory action, the mucous membrane is found to be superficially reduced to a pulpy débris, the separation of which would probably have led to a similar result.[1]

In cases of a severe type, or when the affection of the stomach complicates septic or gangrenous inflammations in other parts, sloughs[2] may form on the mucous membrane, which may have a diameter of from a half to a quarter of an inch, but I have rarely seen them proceed to any depth.

Smaller solutions of continuity may also arise from extravasations of blood, ending in superficial hæmorrhagic erosions. These are, however, much more frequently the result of mechanical congestion than of acute inflammation.

Exsudative inflammations on the surface of the stomach seem also to be rare; I have once or twice seen them in phthisis, and

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[1] See Andral, Clin. Méd. ii. Obs. iv. p. 19.

[2] I am referring to instances entirely independent of corrosive poisons. I have seen them in gangrenous pneumonia, and similar cases are recorded by Recklinghausen (Virch. Arch. xxx.) and by Klebs (Ib. xxxii.).

Sir W. Jenner has observed them in diphtheria, and Rayer[1] says that they have been noticed in cases of croup. Pain and vomiting have been observed in some of these cases, but no special symptoms seem to be attached to this condition enabling a diagnosis to be made.

Another and still rarer affection is that corresponding to the gastritis phlegmonodea of Cullen, which does not as yet appear to have been recognised during life, and which can hardly be included among the class of diseases which have now been described.

The leading symptoms have been acute pain in the præcordial region, together with vomiting, violent fever, and delirium,[2] ending speedily in death.

In cases recorded by Wallmann and Bamberger, the affection of the stomach appears to have been idiopathic, and to have been the only lesion present; but in two others, which occurred in the practice of Oppolzer,[3] it commenced with puerperal fever.

In these instances the chief anatomical character has been suppuration in the submucous tissues of variable extent, sometimes undermining the whole membrane, or perforating it in various places. Bamberger says that the abscesses thus formed may perforate other organs to which the stomach has formed adhesions, but the rarity of these affections renders them objects of pathological, rather than of clinical interest.[4]

Special descriptions of the appearances produced by the different kinds of poisons belong rather to the questions of medical jurisprudence than to the clinical pathology of the stomach. They will not, therefore, be described in this place.

DIAGNOSIS.—The acuter inflammatory disorders of the stomach are usually recognised without difficulty. Some of the severer forms may occasionally be simulated by the neuroses, but the chief points of distinction have been already alluded to.

The slighter forms, especially when occurring in the course of other chronic diseases, and particularly in phthisis, are more liable to be overlooked, the increased prostration and loss of appetite being too frequently referred to the effects of the general disorder. In such cases, symptoms of indigestion of food are not always prominent, and the most valuable indication of the nature of the disease is to be derived from the appearance of the tongue, which instead of being pale, broad, and flabby, as in the atonic state, becomes under these circumstances covered with a milky fur of more or less thickness, through which red papillæ appear. Thirst and constipation are often superadded.

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[1] Dict. de Méd. x. 124.

[2] Bamberger, loc. cit. p. 260. Wallmann, Wiener Allg. Med. Zeit. Decr. 1856.

[3] Wiener Med. Woch. 1851, quoted by Bamberger. They are also described by Rokitanski; by Engel, Lehrbuch Path. Anat.; by Andral, Prec. Path. Anat. ii.; by Albers, Erläuterungen; Dittrich, Canstatt, 1857, iii. 179; Habershon, Guy's Hosp. Rep. 3d Series, ii. 115.

[4] See collection of cases by Reynaud, Bull. Soc. Anat., Paris, 1861.

The diagnosis of the febrile forms from typhoid is also often difficult; and sometimes doubt must continue to exist until after the appearance of the characteristic eruption of the latter. Points which may serve to assist in the diagnosis of the earlier stages are the swelling of the spleen in typhoid, together with tenderness or gurgling in the iliac fossa. The skin also is more frequently dry in typhoid, whereas in gastric catarrh perspirations are common. The temperature also is less elevated in the latter than in the former disease. Herpetic eruptions on the lips or elsewhere, which are common in gastric derangement, are very rare in typhoid. (Niemeyer, loc. cit.)

TREATMENT.—The primary indication in the treatment of recent inflammatory affections of the stomach, is to secure for the organ as complete a rest as it is possible to obtain; and, in the milder forms, a tolerably complete abstinence from food for four-and-twenty hours will frequently do much towards effecting a cure. In severer cases, and where the disorder is more protracted, or when the patient is too weak to bear abstinence, as in the case of infants, much may be effected by nutrient enemata in sparing the stomach, and allowing it the necessary repose. Such food as is taken should, when the disorder is of any severity, be restricted to milk and lime-water, or milk and soda-water, administered in small quantities every two or three hours. In cases where milk disagrees, as it does with some patients, veal or chicken broth, or beef-tea, in small quantities, may be substituted for it.

In the case of infants suckling, the quality of the milk should be carefully examined, and, if the nurse is menstruating, a change is absolutely necessary. If obtained from the cow, the diet of the animal should be restricted to hay or fresh grass. In severe cases it is desirable that the child should be withdrawn from the breast for some hours, and a small quantity of rice-water, or of milk greatly diluted, should be given at intervals. In the case of infants brought up by hand, the milk is to be largely diluted, and the addition of lime-water or of carbonate of soda is very desirable. When cow's milk absolutely disagrees, a change to ass's milk is sometimes sufficient to effect a cure; but this is not always the case, and medicinal treatment is often further necessary. A small quantity of farinaceous food, as arrowroot, sago, or gruel, may often be given with advantage with the milk; it appears to act beneficially by preventing the coagulation of the casein into lumps in the stomach.

In cases of less severity, in older children or adults, the lighter farinaceous puddings may be allowed. Solid animal food is decidedly to be prohibited as long as any nausea or pain at the epigastrium is caused by food entering the stomach; and when the needful rest of body is enjoined, this enforced abstinence may often be protracted during some days with decidedly beneficial effects. The return to a more nourishing diet is always to be effected gradually, and with caution. As the symptoms subside, the patient may be allowed a small quantity of fish, or minced chicken, eaten with bread, but without (at first) any vegetables, and he may gradually proceed to game, or tender mutton, taken once in the day; but it must be remembered that any indiscretion or excess in diet is very likely to bring on a relapse.

Alcoholic fluids are decidedly to be avoided except where great prostration is present. When a stimulant appears to be urgently required, brandy is usually the best form in which it can be given; and in the case of children, and even infants, it may sometimes be advantageously administered, properly diluted, or combined with beef-tea or milk, by the rectum. Champagne also is occasionally found to be advantageous in checking vomiting; but it is less efficacious when this symptom is due to inflammatory action than when it arises from sympathetic irritation, or from the erethism of exhaustion.

Rest of body is equally essential, and in severer cases, and particularly when diarrhœa is present, the patient should be kept in bed until the more urgent symptoms have subsided.

General bleeding is decidedly inadmissible, as the disease ordinarily tends to produce considerable prostration, and any large loss of blood is likely to entail serious consequences, and to retard the recovery of the patient. When pain is continuous, and appears to be unassociated with the presence of undigested food, but to be increased by the introduction of even small quantities of food into the stomach, leeches are often of service. They should not, however, be used in the case of children unless under *very* exceptional circumstances, and in weakly persons the number should be restricted to two or three. If there is evidence of much congestion of the liver, or if hæmorrhoids have been present, they may be applied to the anus; but usually they are best applied to the epigastric region.

Hot fomentations are of value; they should be applied continuously by means of flannel, spongio-piline, or linseed-meal poultices. Counter-irritation by mustard-poultices, or by friction with croton oil, or, in severe cases, with tartar emetic, may be occasionally resorted to with advantage. The warm bath is also frequently of decided service.

Emetics may be administered when the presence of undigested food in the stomach is indicated by cramp-like pain, nausea, ineffectual attempts to vomit, and faintness, but care is necessary in the use of these remedies; and the stronger agents of this class, and especially tartar emetic, or even mustard, are to be avoided (see *ante*), as they have been known to cause great aggravation of the symptoms. The best emetic is a scruple of ipecacuanha, and the emetic action may be aided by large draughts of lukewarm water, or of infusion of camomile; but if vomiting do not occur readily, it is undesirable to repeat the dose, a proceeding which may be followed by very injurious results. It must be remembered that the cramp-like pain originating in the stomach may continue in the intestines when the undigested food has passed into them; and further, as Bamberger has remarked the effects and sensations attributed to the irritant may continue in the stomach in the same manner that the impression of the presence of a foreign body in the conjunctiva may persist long after its removal.[1]

For the condition known as "embarras gastrique," M. Martin Solon[2] has employed ipecacuanha in doses of six or seven grains, given three times in twenty-four hours. In most of his cases the medicine thus given produced bilious stools and vomiting; in several instances a single administration conducted in this manner accomplished the cure; and in others, when a repetition of the medicine was necessary, this was generally effected within three days. In a few cases the disorder was aggravated by this treatment, showing that it is only available within certain degrees of severity of the affection; but the presence of fever by no means contra-indicated its use.

In the milder forms of the affection, even when of some standing,[3] the utility of purgatives is considerable, and has been recognised since the days of Hippocrates.[4] Their immediately beneficial effects were also plainly seen in several of Beaumont's observations,[5] when the redness and aphthous appearance of the stomach, accompanied by a loaded tongue, frontal headache, and sallowness of skin, were relieved by full doses of calomel and aloes.

The best remedies in these cases appear to be mercurials in purgative doses. In the case of an adult and vigorous patient, calomel may be advantageously given in doses of from three to eight grains, and followed after some hours by a draught composed of: magn. sulph. three drachms, magn. carb. two scruples, tinct. jalapæ half a drachm, in aq. menth. pip.; or by castor oil, or the haust. sennæ co., or the decoct. aloës comp. When such active effects are undesirable, the blue pill, with compound colocynth pill and ipecacuanha, often proves very serviceable, and it may be followed by a seidlitz draught, or by any other moderately purgative mixture.

In the case of the acute indigestion of children beyond the period of infancy, when there is fever and griping, or even when the latter is absent, a dose of calomel and scammony, or of hyd. cum cretâ with rhubarb, followed by castor oil, sometimes proves beneficial; but children in this complaint bear purging less than adults, and frequently a dose of castor oil will, if followed for a day or two by suitable care in diet, perform all the service that can be obtained from this class of remedies.

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[1] That undigested food may, however, remain long in the stomach, and be the source of severe pain and discomfort, is shown by two cases related by Sir T. Watson, in one of which a mass of casein, and in another an accumulation of snuff, were vomited, after causing during several days severe gastric pain and disorder. (Lectures, ii. 440.)

[2] Gaz. Méd. de Paris, 1836, No. xvi. I have not resorted to this plan, as I believe, from my own experience, that an active purgation produces equally good effects with less discomfort to the patient.

[3] Andral, Clin. Méd. ii. 186.

[4] "Anorexia, heartburn, vertigo, and a bitter taste in the mouth of a person free from fever, indicate the want of purging upwards and downwards." (Aph. 17, lib. i. sec. iv. Syd. Soc. Trans.)

[5] See loc. cit. pp. 118, 182, 266.

In severer cases, where there is much irritability of stomach, together with frequent vomiting and pain, purgatives by the mouth should be avoided, and they are generally inadmissible when diarrhœa is present, unless there is reason to suspect that undigested food is still retained in the intestines; and the action of the bowels may be procured, if this appears necessary, by aperient enemata.

In rarer instances, when much constipation has preceded or attended the attack, and when vomiting is severe and troublesome, calomel in half-grain doses frequently repeated has been found useful in checking the latter symptom; and a similar indication has been fulfilled by the use of the mixt. magn. sulph. cum magn. carb., repeated at intervals until the bowels have acted freely. Purgatives are also the most efficient immediate remedies for the relief of the sick headaches, though the state of the digestion in which they originate often requires careful attention. Mercurial or saline purgatives are the most efficient in their action, but the activity of the doses should be proportioned to the strength of the patient. Dr. Wood recommends in severe cases the combination of sulphate of magnesia with morphia.

With these exceptions, it is always undesirable to continue the repeated exhibition of purgatives, the effect of which is likely to prove as injurious as a single dose is beneficial, and is liable not only to aggravate the stomach disorder, but to cause an extension of the complaint to the mucous membrane of the duodenum and intestines, if these have not already suffered. Some caution is also necessary in the earlier stages of the febrile forms of the disorder, lest a case of typhoid should be mistaken for one of simple gastric disorder, and serious consequences be entailed on the patient by the administration of a class of remedies which, in the former complaint, must always be regarded as sources of extreme danger.

After the administration of emetics and purgatives, and in the severer cases when these are inadmissible, our main reliance must be placed on sedatives and antacids.

One of the most valuable of the former of these is cold, though it is rarely, if ever, advisable to employ it externally; but sucking small pieces of ice often affords great relief to the uneasiness of the stomach and to the vomiting. Thirst may also be quenched in this manner; but the desire of the patient to drink largely is to be restricted, as distension of the stomach is liable to maintain the tendency to vomiting.

When the vomiting is severe, opium may be administered in full doses with advantage. It is, however, more useful in the severer than in the milder forms of the disorder; and in the latter, which are more appropriately treated by purgatives, salines, and antacids, it is seldom beneficial, and sometimes even proves the reverse.

Hydrocyanic acid is of inferior advantage in checking vomiting in this disorder, but it may be given with occasional benefit in effervescing draughts, containing an excess of alkali.

Bismuth, either in the form of the trisnitrate or subcarbonate, has a peculiarly favourable effect in all the milder forms of inflammatory action; and I have found it more beneficial than any other remedy in the gastro-intestinal catarrh of children. The bismuth should be given in doses of from three to five grains to children, and from ten to twenty grains to an adult; it may be combined with magnesia or hydrocyanic acid, or, when pain or diarrhœa is present, with morphia or tinct. opii.

Carbonic acid is also a valuable agent in relieving pain and vomiting. The mode of its administration has been already alluded to.

In milder cases Vichy, or Seltzer, or soda-water forms a very valuable mode of administering antacids and salines; of the former, which is particularly useful in the later stages of subacute inflammatory attacks, a pint, or a pint and a half, may be taken daily between the meals. The beneficial action of the alkalies is in these cases due in part to their effect in neutralizing the acidity resulting from fermentative changes caused in the food by the unhealthy secretions of the stomach, but I think it probable that some share in their efficacy is also attributable to their direct action on the mucous membrane. Bamberger speaks highly of the value of muriate of ammonia in cases where the disorder threatens to become chronic.

As a general summary of the treatment it may be stated that in the milder forms of the disease one or two doses of a brisk mercurial cathartic, followed by antacids, magnesia, bismuth, Vichy or Seltzer water, and a careful restriction in diet during the attack, will usually bring the disorder to a termination in three or four days. In the severer cases, when pain is felt after food, and vomiting is troublesome, purgatives and emetics are contra-indicated; and repose as absolute as can be obtained for the patient and for the organ, together with opium, ice internally, or small quantities of effervescent drinks, and leeching, fomentations, or counter-irritation to the epigastrium, are the indications principally to be relied on for obtaining a cure.

A weakened state of the digestion, entailing a liability to fresh attacks, often continues after the acuter symptoms have subsided; it is attended with a feeling of languor and of inability for active exertion, and not unfrequently with flatulence or occasional heartburn, or with some other of the symptoms of atonic dyspepsia. This state requires great care in treatment, and the use of the so-called bitter tonics demands especial caution, as their untimely use will often perpetuate a chronic form of subacute inflammatory action.

The general rules laid down for the treatment of atonic dyspepsia are those most applicable in these cases; and attention to diet is to be pursued with additional caution, which is to be especially extended to vegetables and fruit. Exercise should be moderate, and should not be pushed beyond a degree sufficient to cause a slight and healthy feeling of fatigue. Chills should be carefully avoided; for patients in this condition are as liable to "take cold" in the stomach as others, in whom the respiratory system is weakened, are to attacks of bronchitis. Alcoholic stimulants are only to be used in great moderation—dry sherry (the Manzanilla or Amontillado being the best), or weak brandy and water, or claret and water, are the most suitable forms for their administration; malt liquors, and also the use of coffee, are to be decidedly forbidden. If all signs of irritation have disappeared, while those of atony continue, and if the deficiency in the appetite indicates the use of bitters, calumba and nux vomica are those most applicable; these may generally be advantageously combined with the mineral acids, and should be given immediately before food is taken.

Pespine, also, often proves very serviceable at this stage.

If anæmia be present, the milder ferruginous preparations are to be employed in the manner indicated under the head of Atonic Dyspepsia.

Care and patience are at all times necessary in cases of this description. The tendency is to a cure, if it be not interfered with by undue haste on the part of the patient to regain strength, by taking food in excess of the digestive powers of the stomach, or by the use of stimulants beyond a degree which the weakened tissues are able to support without their normal action being perverted to inflammatory irritation.

Purgatives, which the sluggish condition of the bowels may sometimes appear to render necessary, are also to be used with great care. Aloes is usually the best form that can be selected; but the use of enemata is to be preferred. In children, small doses of rhubarb and soda, or of the domestic Gregory's powder, are often decidedly advantageous; or friction with aloes may be employed on the surface of the abdomen.

In infants, small quantities of magnesia will often answer all indications in this respect; but in them the tendency to diarrhœa is more marked than in adults, and purgatives are seldom necessary.

In older children and in adults, the action of the bowels is often facilitated, and irritability of the gastro-intestinal membrane greatly relieved, by wearing over the abdomen, either during the day or night, a compress wrung out of warm or cold water, and protected by a piece of mackintosh. It should be changed three or four times in the twenty-four hours.

Change of air and scene, as spoken of under the head of Atonic Dyspepsia, is sometimes necessary in weakly patients to complete a cure.

V.—CHRONIC GASTRITIS.—CHRONIC CATARRH OF THE STOMACH.

SYNONYMS.—Chronic Inflammatory Dyspepsia, Morbid Sensibility of the Stomach (Johnson); Gastrite Chronique, Fr.; Chronisches Magen Catarrh, Germ.

DEFINITION.—A disorder of digestion, the symptoms of which consist in the imperfect assimilation of food, associated with physical and moral depression, with irregular appetite, thirst, and slight degrees of pyrexia, and with impairment of the general nutrition, attended in some cases by gastric pain, by the vomiting of mucus, and by various alterations in the urine, and which depend on chronic inflammatory conditions of the stomach, revealed by congestion, pigmentation, and thickening of the mucous membrane, and by atrophy and degeneration of its secretory glands.

This disease embraces a large number of the cases of obstinate chronic dyspepsia, and includes, in the author's opinion, many of the disorders which have been described as irritative dyspepsia, and even some which have been ranked among the nervous disorders, and especially those forms which have been regarded as resulting from "morbid sensibility"—a condition which in mucous tissues is much more commonly the result of minor degrees of inflammatory action than of mere derangements of innervation. Confusion has also been introduced into the nosology of this disorder by the inclusion under this title of cases of ulcer and cancer,[1] and especially of the former.

THE ETIOLOGY of this affection is somewhat complex, since the disorder may either exist *ab origine* in a subacute form, perpetuated by the persistence of its exciting causes, or may remain as an effect of an acute attack, which has weakened the nutrition of the mucous membrane, and has thus induced a liability to minor degrees of inflammatory action from slight causes. It is therefore difficult logically to define in all cases the limits between the predisposing and the exciting causes, since many of each class act apparently in both directions.

The disease appears to be to a certain degree hereditary, but no special etiological influences can be attributed either to sex or age, as it is common at all periods of life.

The impaired condition of the digestive powers which has been described under the head of Atonic Dyspepsia induces liability to inflammatory disorders of the stomach, and hence the causes of this state may all act as indirect predisposing causes of the irritative forms, both of the acute and of the chronic kind.

Among the other constitutional diseases which appear to involve a special liability to this affection—though often acting apparently in diverse manners—may be mentioned scrofula, phthisis, gout, albuminuria, and diseases tending to disturb the portal or abdominal circulation, such as emphysema, heart disease, and, *à fortiori*, cirrhosis of the liver, or other conditions affecting the circulation through this organ. The following table exhibits the principal diseases with which I have found either acute or chronic catarrh in 100 stomachs examined:—

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[1] Cf. Andral, Clin. Méd.

Acute Catarrh.	No. of Cases.	Chronic Catarrh.	No. of Cases.	Acute and Chronic Catarrh combined.	No. of Cases.
Pneumonia, Acute	2	Tubercle of Lungs (uncomplicated)	4	Tubercle of Lungs (uncomplicated)	2
——— Chronic	1	Tubercle of Lungs, Tubercular Peritonitis	1	Tubercle of Lungs, Morbus Cordis	1
Variola	2	Tubercle of Lungs and Intestines, Bronchi-Ecstasis, Morbus Cordis	1	Tubercle of Lungs, Tubercular Pericarditis, old Valvular Disease of Heart, Liver Fatty, Kidneys Granular	1
Puerperal Peritonitis, with recent Bright's Disease	2	Tubercle of Lungs and Intestines, Liver Fatty	1	Tubercle of Lungs, recent Bright's Disease	1
Perimetritis Puerpera, with recent Bright's Disease	1	Tubercle of Lungs, Morbus Cordis, Granular Degeneration of Kidneys	1	Morbus Cordis	2
Phlebitis, from various causes, associated with first stage of Bright's Disease	2	Tubercle of Lungs, Morbus Cordis	1	Morbus Cordis, Cirrhosis of Liver, recent Pneumonia	1
Endo-Pericarditis, first stage of Bright's Disease	1	Capillary Bronchitis, Morbus Cordis	1	Pneumonia (uncomplicated)	1
Suppurative Parotitis	1	Chronic Bright's Disease, Fatty Liver	2	Pneumonia, Phlebitis	1
Cholera; Kidneys in first stage of Bright's Disease	2	Morbus Cordis, Gangræna Pulmonum	1	Morbus Cordis, Gangrene of Lung	1
Typhoid Fever	1	Morbus Cordis, Caries of Pelvic Bones	1	Pneumonia, recent Bright's Disease	1
Morbus Cordis	1	Melanosis of Lung, Pleurisy	1	Puerperal Fever	1
Morbus Cordis, Capillary Bronchitis	1	Cystic Disease of Ovaries, Peritonitis	1	Typhoid Fever	1
Tubercle of Lungs	1	Abscess in Œsophagus	1	Delirium Tremens	1
Tubercle of Lungs and Tubercular Peritonitis	1	Hernia	1	Cirrhosis of Liver	1
Diabetes	1	Drunkard. Other pathological conditions not noted	1	Suppurative Parotitis, Kidneys Fatty	1
Carcinoma Ventriculi	1				
Total	21	Total	19	Total	17

One remarkable fact which appears from this table, especially in the contrast between cases of recent and chronic catarrh, is the greater proportionate frequency with which the former is associated with acute, and the latter with chronic, inflammatory affections of other organs; and it would seem as if the same exciting cause not unfrequently sufficed to produce similar changes in the ultimate structures of many organs simultaneously.

The tendency of diseases obstructing the venous circulation to induce chronic catarrhal conditions of the stomach is also well illustrated in this table; but the remarkable frequency with which they are associated with phthisis is especially apparent, having been present in 28 per cent. of the whole number, or in sixteen out of thirty-one, or nearly one-half, of the tubercular cases examined.[1]

Of the more direct exciting causes, among the most frequent must be placed either an habitual excess in eating, or a constant use of food that disagrees; especially by persons whose general health and digestive power are below the healthy standard: by this means a series of minor attacks of indigestion of an irritative type are excited, which at times alternate with acuter forms of "embarras gastrique," from which such patients are continually liable to suffer.[2] It cannot be too strongly insisted on, that the tendency of undigested food is always to give rise to gastro-intestinal irritation, whether the cause of the indigestion reside in the stomach, or in the quality or excessive amount of the food or drink, or in some of the other accidental conditions mentioned among the general causes of dyspepsia,—of these deficient insalivation or imperfect mastication, or the habits of mental or physical work after meals, are among the most frequent.

The habitual use of spirits, particularly when undiluted, or when taken to any amount that can be considered excessive, is also an almost unfailing cause, when long continued, of chronic inflammatory changes in the stomach; and it is seldom in cases of this nature that some of the post-mortem changes hereafter to be mentioned cannot be discerned.[3]

Chronic or subacute catarrh of the stomach is also a very frequent accompaniment of cancer and ulcer of this organ, as well as of pyloric obstruction. The question of the mode of its causation in the two former of these diseases is one of no little difficulty, but it is probably to its agency that many of the derangements of digestion observed in their course are attributable.

Medicinal causes deserve also some consideration, and especially the abuse of stimulants and tonics or purgatives in many of the forms of atonic dyspepsia; nor can other remedies be absolved from the onus of having occasionally produced these effects, which have been attributed to the prolonged use of arsenic and bichloride of mercury, and occasionally of cubebs and copaiva.

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[1] The author's more recent observations, though not capable (from peculiar circumstances) of being framed statistically, have fully confirmed his opinion of the etiological influence in this direction of these classes of disease; and he has been increasingly struck with the frequency of the co-existence of the anatomical conditions to be described, as found in chronic catarrh, with cirrhosis of the liver and with granular conditions of the kidney, to which they bear the strongest resemblance. Dr. H. Jones's observations also give some support to this view, for, in twenty-three cases of catarrh of the stomach, he found tubercles in four and disease of the lungs in eleven more, while in eight there was disease of the kidneys, and in three disease of the heart.

[2] Broussais (Lec. Phleg. Gastriques, p. 183) says that the causes which in adult age perpetuate a chronic irritation would, in the earlier periods of life, have produced an acute attack.

[3] The average ratio, in persons who have indulged in these excesses, of deaths from the digestive organs (including disease of the liver) to deaths from all causes, according to Neison's researches, amounts to 12·47. ("Vital Statistics," quoted by Parkes on Hygiène, p. 299.) I believe that the amount of injury to the stomach is much underrated, since changes in this organ often escape observation, and are rarely directly the cause of death.

There are some cases of syphilis recorded, accompanied by symptoms closely resembling those characterising this group of diseases, and which have been cured by the administration of mercury. The evidence of their nature is incomplete, but, pending their complete elucidation, I am inclined to believe that, judging of the effects of this poison on other tissues, such disorders are most suitably classified among the inflammatory affections of the stomach.

Lastly may be mentioned, mechanical irritants of all kinds.[1]

THE SYMPTOMS of this disorder are primarily those of indigestion of an aggravated kind, but they are often varied, and very irregular in their course; nor are they always united in one case, some being at times more prominent than others. It is of this class especially that the remark made with regard to the general symptoms of indigestion will be found to be true, that the condition may often be revealed by the general state of the system, or by impairment in the functions of some other organ, rather than by symptoms appreciable by the patient himself, as truly proceeding from the deranged stomach.

In many cases there are at times intervals of almost complete immunity from apparent dyspepsia, but these are seldom complete, and are speedily followed, often with no apparent cause, by returns of the old symptoms, and with exacerbations of an acuter kind, after the slightest indiscretion in diet; and this irregularity is of no small value in distinguishing the nature of the complaint, having its parallel in most of the chronic inflammatory affections of other mucous membranes.

Those referable to the stomach are a sense of weight, oppression,[2] distress, or undefined uneasiness of the epigastrium, ensuing after meals, and often associated with distension from flatulence, which sometimes may be very considerable; a sense of tightness and constriction at the sternum, or a feeling of fulness in the pharynx and œsophagus; and in other cases an uneasy, ill-defined sensation of discomfort is felt in the dorsal region between the scapulæ, though rarely also in this situation amounting to the acuter pain met with in ulcer and cancer. The sensations complained of generally commence within half an hour to an hour after taking food, and continue more or less during the whole period of digestion. Other discomforts follow at a later period, such as intestinal flatulence, and borborygmi, together with an increased sense of distension and oppression, especially felt in the right hypochondriac region. Sometimes these uneasy sensations, when accompanied by acidity, are relieved by taking more food; but this alleviation is frequently only temporary, and is followed by an increase of the original discomfort.

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[1] See a case, of pins swallowed by an hysterical woman. (John Marshall, Med.-Chir. Trans. xxxv.)

[2] *Barre* of the French writers, described "as if a bar were pressed across the epigastrium, or base of the chest."

The ingestion of food is rarely, if ever, a cause of immediate pain; and this is not, indeed, a prominent or (with any severity) a frequent symptom, though it may occasionally arise if much mucus is secreted, or when flatulence is present.

Tenderness on pressure is not usually a marked symptom, though some degree of it often exists. Heartburn and acidity are often very annoying, but they are by no means constant.

Nausea, to a certain degree, is not unfrequent, but it is seldom distinctly felt; and vomiting is rare, except in certain special forms, associated with either albuminuria, congestion, the dyspepsia from drink, and occasionally with phthisis.

The appetite presents considerable variations. It is capricious, and generally it is diminished, though not, as a rule, to a marked degree. Eating is, however, soon followed by a sense of satiety; and the feeling of discomfort and fulness at the stomach which often ensues, even during a meal, serves at once to check the desire for food; though if this be neglected, and a full meal taken, the symptoms are usually increased in severity. In other instances there is the same sense of sinking, and of craving for food, which is observed in some of the neuroses, and which must in these cases be considered as a perverted sensation arising from the condition of gastric irritation; though it should be borne in mind that nervous erethism may at times complicate this complaint, and introduce much complexity among the symptoms observed.

Thirst is a very prominent and distinctive symptom, and it is hardly ever absent in cases of inflammatory irritation of the stomach, whether this be acute or chronic. It exists during meals, but is most marked in the intervals, when the patients have an extreme craving for fluid; and this is particularly felt in the evening, if the chief meal has been a late dinner. The feeling is not only one of thirst, but a sense of languor, oppression, exhaustion, or internal heat is often combined with it, which is relieved by drinking, and especially by cold fluids; but in some cases even these give distress, and warm drinks, especially tea, are eagerly taken, though often in the latter case only to be followed by increased discomfort, acidity, and flatulence.

The breath is often heavy and offensive. There is very frequently a bad taste in the mouth, which is ordinarily most marked on first rising in the morning. The gums are spongy, red, swollen, often retracted from the teeth, and inclined to bleed, and the saliva and buccal mucus are occasionally acid. An excessive flow of saliva is not uncommon, and is particularly observable at night, when it may escape from the mouth during sleep, wetting the pillow. The lips tend to become dry and cracked, and the fauces are liable to erythematous inflammation, with slight superficial ulcerations. The pharynx also may be the seat of a granular inflammation, associated with excessive secretion of a tenacious mucus, which is a source of great annoyance and discomfort to the patient.

The conditions of the tongue present some variations; but as these are valuable aids to diagnosis, they deserve to be especially dwelt upon.

(*a*) If associated with distinct atony, the tongue may be broad, somewhat pale, and flabby, but the papillæ generally are enlarged—this being most apparent in the fungiform papillæ on the tip and edges, which are also redder than natural, and there is a thin white fur over the surface. Sometimes, however, this fur may be present when the papillæ, though enlarged, are pale.

(*b*) In the more distinctly irritative forms, and especially in children who have any signs of scrofula, and in phthisical adults, the whole organ is redder than natural, and may be of a bright florid colour, and even raw-looking; it is often pointed at the tip, which, together with the sides, presents an extreme degree of injection, and the papillæ stand out as vivid red points. There may be at the same time a coating of variable thickness along the dorsum. This form is frequently associated with aphthæ, especially at the tip, and sometimes on the inside of the lips; or with painful spots on the tongue, which are found on close examination to be papillæ slightly abraded.

(*c*) In older persons, and particularly in those in whom the dyspepsia is the result of excessive or hurried eating, the tongue, while presenting some degree of enlargement and redness of the papillæ at the tip and edges, is often uniformly covered throughout the greater part of its extent with a thicker fur, sometimes whitish, and occasionally of a browner tint, which more resembles the coating attending the acuter attacks, and which patients recognise as a symptom of "biliousness."

(*d*) Lastly, it must be mentioned, that in cases which I cannot but regard, as far as my experience extends, as being exceptional, though more common where the catarrh of the stomach is secondary to local causes of congestion from venous obstruction, the tongue may present very little deviation from the natural condition, though occasionally, even in these, transitory formations of a white fur on the dorsum may be observed on repeated examinations.

Some of the intestinal symptoms have been already alluded to. In addition it must, however, be noted, that constipation is often obstinate; it is frequently associated with much uneasiness in the rectum, and it greatly increases the general feelings of oppression, malaise, and languor. The stools may be dry and scybalous, and are not unfrequently coated with a considerable amount of tenacious mucus, which may form casts of portions of the intestinal canal. Occasionally they are passed with tenesmus and straining; sometimes they present thin flattened bands; and they are usually accompanied or preceded by the escape of flatus. They are generally pale, both in this state and in another occasionally met with, when the constipation is less marked, and when one or two large, pultaceous, often offensive, and sometimes frothy, motions, containing considerable quantities of undigested food, are passed in the day—when there are often griping pains in the abdomen, and a liability to severer attacks of colic. In other cases the two conditions alternate, or slight causes may give rise to transitory attacks of diarrhœa, which may afford temporary relief, but are commonly followed by an aggravation of the intestinal flatulence and of the general discomfort, and are often attended with increased signs of irritation on the tongue and with the production of aphthæ, as before described.

Piles are a not uncommon complication of this state, even when evident disease of the liver is absent.

The cutaneous surface shows various indications of the perverted general nutrition of the body. It is often dry and harsh, sallow, earthy, and wrinkled; and at times, after slight indiscretions of diet or without assignable cause, patients are liable to suffer from eczematous or impetiginous eruptions, which may be followed by a perceptible alleviation of the symptoms.[1] Children are more liable to suffer from these, and occasionally from herpetic eruptions, than adults; but gouty patients, and those who drink excessively of malt liquors, are occasionally subject to them. A vinous tint on the malar bones and nose is also by some writers attributed to the disturbance of the stomach.[2]

The hair tends to become dry, harsh, and prematurely grey; sometimes it is lost in considerable quantities when acuter exacerbations supervene. The nails are often furrowed, and have a tendency to split. In children who suffer much from irritative dyspepsia during the second dentition, the teeth are often irregular, with thin enamel, and are crenated at the edge, while the anterior and, to a less degree, the posterior surfaces are marked by vertical depressions or sulci.[3] In adults the teeth suffer from premature caries, often erroneously attributed to the use of mineral acids given for the cure of the complaint, but more commonly due to impaired general nutrition, and especially to the spongy condition of the gums and to their retraction above the enamel.

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[1] A case of this kind lately came under my notice, of a young lady whose father is liable to gout, and who had for years been liable to an aggravated form of irritable dyspepsia, but whose symptoms almost entirely disappeared for many months since the eruption of an eczematous rash on her face. Trousseau (Clin. Méd. ii. 1862, p. 280) has noticed that in dyspeptic patients who resort to sea-bathing a febrile condition, followed by urticaria, frequently results, and that the eruption on the skin is followed by a great relief to the symptoms of the stomach. Allusion has been already made to the occasional concurrence of this disorder with some of the forms of acute indigestion, and a somewhat similar instance is also mentioned by Schmidtmann (loc. cit. iii. 225):—"Novi mulierem in qua herpes faciei cum cardialgia alternabat; extante in facie herpete a cardialgia vacabat; eo disparente, extemplo duris torquebatur ventriculi doloribus."

[2] Rayer, Dict. de Méd. x. 156; Chomel, ib. x. 92.

[3] This condition of teeth is very common among children to whom "grey powder" has been administered at the time of the second dentition; but I have frequently observed it, independently of this cause, where there has been much dyspeptic disturbance at this period. In a third class of cases it is hereditary, appearing when the digestion is good; but it is not unfrequently associated with scrofula.

Emaciation is almost constantly observed when this disorder has persisted for any length of time, though it is not early in its appearance, unless the patient has been previously out of health, or the disease is severe; but a gradual loss of flesh and strength is an almost constant symptom, and one that should, in the absence of other signs of disease, cause a special attention to be directed to the state of the digestive organs.

Coldness of the extremities is a very common symptom, and it is not unfrequently attended with flushing of the face and oppression of the head. Patients of this class are almost always chilly. They are liable to slight rigors and to suffer much from changes of temperature, which are often followed by an aggravation of their sufferings.

Febrile reaction of a slight type, preceded by rigor and malaise, is very common. It often appears to be directly associated with the taking of food, or of alcoholic stimulants, but in other cases it seems to have a special tendency to exacerbation in the evening; sometimes returning with such regularity as to have given rise to the suspicion of a malarial cause, and to have led to the ineffectual use of quinine.[1] The skin becomes hot and dry, especially in the feet and hands; but in other cases these may be cold, while a great sense of heat is complained of in the trunk and head. It occasionally occurs at night, and then is often followed by copious perspirations during sleep.[2]

There may be at times an icteric tint of the conjunctivæ, but this is not observed with any considerable frequency. Slight attacks of ocular conjunctivitis, sometimes attended with phlyctenæ, are by no means uncommon.

Slight catarrhal affections of the air-passages are also not unfrequent. Those of the fauces and pharynx have been already alluded to; but the same condition may invade the larynx, giving rise in some cases to injection, with relaxation of the vocal cords, and thus causing dry cough, or hoarseness and huskiness of the voice; or the affection may extend deeper, and be the source of a muco-purulent secretion, which is often aggravated during the febrile accessions which occur after meals, and especially after wine has been taken, and may give rise to cough coming on at these periods.

Dyspnœa, and a desire to sigh, are very frequently complained of. They are sympathetic symptoms common to all forms of indigestion, but are very marked in the variety now under consideration.

A very interesting and important question connected with this subject is the connexion of these disorders to the causation of phthisis.

By some authors[3] disturbance of the digestion has been considered to be an immediate cause of the development of pulmonary tubercle; while by others the relation of the two conditions has been considered as accidental; or it has been held that the irritative dyspepsia so often observed in phthisis is secondary to the tuberculizing process in the lung.

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[1] Chomel, Des Dyspepsies, p. 79.

[2] It may be well here to recall the aphorism of Hippocrates in relation to the causation of many of these forms, which has been already alluded to: "A copious sweat after sleep, occurring without any manifest cause, indicates that the body is using too much food." (Aph. 41, sec. iv.)

[3] Wilson Philip, Mr. Hutchinson (Med. Times and Gazette, 1855). The dyspepsia

It would appear, however, to the author that in cases where long-continued irritative dyspepsia has preceded disease of the lung, the latter has required for its production some other exciting or predisposing causes than the simple impairment of nutrition produced by the mal-assimilation of the food. In the majority of instances where they co-exist, both the gastric and pulmonary disturbances have either appeared to be due to an unhealthy constitutional condition, and have been developed and advanced almost *pari passu*, each accession of pulmonary disorder with pyrexial disturbance being associated with a fresh attack of gastric catarrh, which has tended to become chronic; or the derangement in the stomach has been secondary to that in the lung, and caused either by pyrexial conditions, or by impairment of the general health, or possibly by reflex irritation.

The urine, in most of the chronic inflammatory affections of the stomach, is more constantly affected, in various ways, than in the other forms of disturbance of digestion before noted. The most common of the changes presented are the deposits of urates, earthy phosphates, and oxalates. None of them is peculiar to this form, and moreover urine of high specific gravity, and characterised by the deposit of urates, is almost invariable in the acuter stages of inflammatory dyspepsia.

Earthy phosphates, with urine sometimes of a high, and sometimes of a low, specific gravity, and often alkaline and cloudy on emission, are very common. The specific gravity depends, in some measure, on the amount of fluid taken with the meal preceding the period at which the urine is passed; and thus it is commonly low in the morning after breakfast, and generally higher in the evening, when the exertions of the day and the food taken have probably increased the amount of urea. By some authors this condition of cloudy, alkaline urine, which often becomes iridescent on standing, has been referred to duodenal indigestion,[1] this idea having probably arisen from the fact that this deposit in the urine frequently corresponds with the period at which the food passes into the intestines, and with the aggravation of the symptoms of malaise and flatulence which often occurs at that time. The author is inclined to believe that the alkalescence of the urine in these cases may be in part due to the defective secretion of the liver and pancreas; of the former of these we have evidence in the pale, clayey stools, and also some presumptive proof in the great increase of flatulence which occurs in the intestines. The fact that these deposits often alternate with urates, and sometimes with oxalates, lends a further support to this view; since the latter seem

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preceding phthisis has been attributed by Dr. Hughes Bennett and Mr. Hutchinson to a dislike to and mal-assimilation of fatty substances.

[1] Cf. Yeats, Some Observations on Duodenum, Med. Trans. Coll. Phys. 1817, p. 351, and Mayer, Krank. des Zwölffinger Darms, p. 10.

to be invariably associated with a faulty assimilation or metamorphosis of protein and saccharine matters,[1] and may probably depend on the abnormal condition in which these enter the blood after the imperfect intestinal digestion caused by the deficiency in supply of those secretions.

The deposit of urates is often associated with the febrile heat complained of after meals, but the same symptom has been noted in connexion with both oxalic and phosphatic deposits.[2]

The nervous system participates markedly in the general disturbance.

Languor, lassitude, pains in the trunk and limbs—the latter sometimes dull and aching; sometimes, when in the scapular region, severe and lancinating; at others directly affecting the muscles, and simulating conditions of chronic rheumatism,—a feeling of inability for exertion, especially marked after meals, and often felt on rising in the morning, irritability or excitability of temper, intellectual depression, loss of judgment and of the reasoning powers, and of memory, characterise this state. Hypochondriasis occurs also more commonly in connexion with this condition than with almost any other form of dyspepsia. Fear, timidity, anxiety; despondency to such a degree that, "in a merchant surrounded by affluence, apprehensions of impending beggary often embitter the moments that are free from the excitement of business; in the mechanic, unfounded ideas of immediate loss of employment, and visions of the interior of a workhouse, are generally present."[3]

Headache and a feeling of tension are frequently present; but the sick headache is not so common in advanced forms of the disease, (unless under the supervention of acute attacks of indigestion,) as in the simple atonic or acute forms; the feeling being generally rather one of fulness, or of dull pressure, in the occipital or frontal regions. Vertigo is occasionally met with, especially when irritation has supervened on the atonic form; but with this exception it is, comparatively speaking, rare in this variety.

The expression is anxious and careworn, and, in conjunction with the emaciation and sallow tint of skin so commonly present, gives to the individual a look of premature age.

Extreme degrees of sleeplessness are very common; or when, after hours of restlessness, sleep is at length attained, it is disturbed by dreams and nightmare, and is sometimes associated with nocturnal emissions.[4]

The heart's action is often irregular, and easily excited to painful palpitation on slight exertion, conjoined with which there may be at times some irregularity of action. The pulse is accelerated during the febrile movement following digestion ; it is then full and compressible ; at other times it becomes weak and slow, in proportion as the patient loses strength by the continuance of the dyspepsia.

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[1] Parkes, On the Urine, p. 225; Roberts, loc. cit. p. 43; Golding Bird, p. 159.

[2] Golding Bird, loc. cit. pp. 244, 291. Phosphatic urine and oxaluria may, indeed, occasionally occur when the condition is one of simple atony or neurosis, but it is probable that when they are persistent, or of any severity, some conditions of organic irritation, tending still further to impair the digestive power, are almost invariably present.

[3] Ibid. p. 308. This mental state, so graphically described by Dr. Bird as occurring in connexion with phosphatic urine, may be found in conditions of irritative dyspepsia, when these changes in the urine are not at the time present.

[4] Chomel.

In the foregoing sketch a description has been given of the leading symptoms which correspond to cases where the anatomical characters, hereafter to be described, are found ; but the whole of this group does not invariably appear simultaneously. The course of the disorder is also modified by the various etiological conditions under which it occurs, bringing certain symptoms into greater prominence than others, and also by its occasional complication with some of the neuroses, to which reference has previously been made, and which are among the chief sources of obscurity in the diagnosis of the severe forms of both affections.

The most typical examples of the disease, as affecting both the digestive organs and the nervous system, are those where irritative dyspepsia has supervened in a debilitated constitution, and has been preceded by the symptoms of atonic dyspepsia. They are yet more marked if in such a constitution there is any taint of hereditary gout, which may not have been revealed by a distinct attack, but where the emaciation, weakness, and hypochondriasis are familiar to all who have had opportunities for observing these complaints.

In the scrofulous forms in children, the attention of the parents may be directed only to the pallor, weakness, and gradually progressing emaciation of the patient, and the evil is constantly aggravated through ill-advised attempts to improve the nutrition by forcing increased nourishment on a stomach already incapable of digesting the normal amount. Phthisical dyspepsia, on the other hand, is often painfully complicated by the diarrhœa proceeding from ulcers in the intestines, but which possibly in some degree aids in preventing the oppression and hypochondriasis which so frequently attend the constipation presented by some of the other varieties.

There yet remains a group of cases where vomiting constitutes a more prominent symptom than in those which have hitherto been passed in review, and where there occurs a profuse secretion of mucus, and which stand in the same relation to the forms in which this is not so apparent as a bronchorrhœa, or leucorrhœa, or nasal catarrh occupy to the drier forms of inflammation of the mucous membrane from which those fluxes may proceed. The cases where gastrorrhœa is a prominent symptom are ordinarily those of congestion of the stomach from pulmonary, cardiac, or hepatic disease, and of dyspepsia in habitual drunkards, in which the last-named cause of congestion often, I believe, plays a prominent part; and to these must be added many cases of albuminuria.[1]

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[1] Bernard's experiment has shown that after extirpation of the kidneys a continuous secretion takes place from the stomach without any necessary alteration of its mucous membrane ; but in cases of longer duration of Bright's disease pathological observations

Sometimes the vomiting in albuminuria occurs on first rising in the morning, when it is occasionally relieved by food:[1] under these circumstances it is possible that it is of cerebral origin, and is caused by the disturbance of the nervous centres through the poisoning of the blood: but in a large class it takes place within half an hour to an hour after food has been taken. Pain, though sometimes present, and occasionally severe, is, however, but rarely complained of; and though much acidity is sometimes present, the reaction of the vomited matters may be at other times alkaline: but the cause of this difference has not, as far as I am aware, been made the subject of special observation.

In the dyspepsia of drunkards the vomiting of mucus is often one of the most prominent symptoms. It usually occurs in the morning, and is easily excited by slight stimuli. During the rest of the day there may be comparatively little disorder in the stomach, though acidity and flatulence are sometimes present, and the appetite is often greatly impaired. In other cases a painful sense of sinking is experienced at the epigastrium, together with a craving for the accustomed stimulant, which too often replaces all desire for food. The disturbances arising from this indulgence affecting the nervous system, the ascites and sallow skin, the icteric tint of the conjunctivæ and the signs of cirrhosis, belong more properly to other sections of clinical medicine and pathology.

In some cases, however, a symptom common to it and to other forms of congestion occurs, viz. hæmatemesis, which may occasionally be profuse, and return with such frequency as to threaten life, and to reduce the patient to an extreme degree of anæmia. The severer forms are most frequently associated with cirrhosis of the liver, causing obstruction of the portal vein; but minor degrees of the affection often appear in conjunction with disease of the kidney. It is very probable, judging from the results of post-mortem observation,[2] that hæmorrhage not unfrequently takes place without being disclosed by the blood vomited, the matters brought up being chiefly alkaline mucus (sometimes considerable in amount, and which may here and there only have a coffee-ground tint), bile, or altered food. Sarcinæ are occasionally found in the vomited matters of the whole of this group of cases.[3]

Pain in these cases is a symptom which is variable in the frequency of its occurrence; it is often complained of after each meal, but is seldom, if ever, of marked severity. Flatulence is almost constant. Acidity is common, but is not comparatively so frequent. The

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have convinced me that the stomach seldom fails to exhibit signs of subacute inflammatory action (see also Rayer, Mal. des Reins, ii. 347). These anatomical changes are probably due to the continuance of the unnatural secretions.

[1] Christison, Granular Degeneration of the Kidneys, p. 96.

[2] See Handfield Jones, "Stomach," p. 91, where grumous blood was found in the stomach after death, none having been vomited during life. I have met with several such cases.

[3] For the more special description of the nature of the matters ejected, the reader is referred to the chapter on Vomiting as a symptom.

progress of the disorder, in other respects, depends much on the complications with which it is connected.

Pathology.—The changes which accompany the more marked forms of this disorder are tolerably characteristic. Many of them are apparent to the naked eye; others are only disclosed by microscopic examination. The most distinctive of these are alterations in the vascularity of the mucous membrane, changes in its colour, increase in its thickness and resistance, occasional increase of the lymphatic elements in the intertubular tissue, and various forms of degeneration of the glandular and other structures.

The degree of post-mortem vascularity, however, presents the same difficulties as a criterion as were mentioned in the description of the appearances observed in the acuter form; and even congestion from obstruction, sufficient to give rise to hæmorrhage, may leave no distinct traces in the mucous membrane after death. Often, however, the long continuance of distension of the vessels produces an amount of dilatation, which, when combined, as the affection frequently is, with exacerbations of an acuter kind, gives greater post-mortem evidence of hyperæmia than is found in the cases when inflammatory action has been of shorter duration.

Where much congestion has been present, the hæmorrhagic erosions previously alluded to are also very common,[1] and they depend on an extravasation of blood in the substance of the mucous membrane, which results from capillary rupture. They seldom exceed two or three lines in diameter, and they are generally superficial; but they may be sometimes seen extending through the whole depth of the mucous membrane. In some places the tissue may be seen still infiltrated in patches, where the blood has been effused, without detachment of the softened surface; but in others there are seen little pits or depressions with a blackened base, and with sides still infiltrated with blood, which, on microscopic examination, is found to occupy the tubules, staining their epithelial contents. They may possibly in some cases be the source of pain, but, when small, they appear to have little other pathological significance.[2] Vascularity may be found in any part of the mucous membrane; but changes of colour and thickenings of the tissue are more common in the pyloric portion, as also are many changes in the glandular tissue hereafter to be described.

The most characteristic change in colour is an ash-grey pigmentation,[3] which, when closely examined, is found to depend on minute black specks scattered closely over the surface of the membrane. It is generally most marked in the pyloric half of the stomach, though traces of it may occasionally be met with near the cardia. When examined with the microscope, these spots are found to depend on pigment derived by imbibition from the hæmatin of the blood, and deposited in a minutely granular form in the cells of the connective tissue between the tubes, and sometimes in the epithelial contents of the latter. It is most commonly met with when mechanical causes of congestion have co-existed with catarrhal changes, and requires probably, as an antecedent condition for its production, the rupture of capillaries in the superficial layers of the membrane; and it is very commonly associated with similar changes in other parts of the intestinal tract. But though its presence is a valuable indication of the nature of the causes in which it has had its origin, its absence by no means excludes the diagnosis of past inflammatory action, of which other and independent evidence can be found in the consistence of the membrane and changes in the glands, and which, though often associated with pigmentation, can also frequently be discovered when this is absent.

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[1] Willigk, Prager Viertel-Jahreschrift, vol. 51, gives their frequency as 1·8 per cent. of all the bodies examined. I know of no statistics mentioning their relative frequency to causes likely to produce these extravasations.

[2] Larger extravasations appear, however, sometimes to serve as the origin of the chronic ulcer of the stomach.

[3] Andral described a milky-white colour of the membrane as characteristic of chronic inflammation. There is generally a certain degree of opacity induced in this condition; but unless an acute affection should have supervened before death, I do not think that it is common in this disease, except in spots resulting from fatty degeneration of the glands,

Thickening and induration of tissue are an almost uniform result of chronic inflammatory action in the mucous membrane of the stomach. This may at times acquire an extreme degree of firmness and resistance,[1] tearing with great difficulty, and being capable of being stripped from the submucous tissue in large pieces. There may be sometimes a slight degree of softening of the surface, when recent inflammation has supervened on the chronic form, but it does not usually extend sufficiently deeply to affect the general characters of induration which are so characteristic of this state. These changes depend on an increase of the interstitial tissue between the glands, which is often associated with atrophy of the latter; but it may also exist when this secondary change has not ensued.

Enlargement of the solitary glands, together with an increase of the lymphatic interstitial growths between them, is common, though not constant, in this form.

The mammillation considered by Louis[2] as a constant sign of inflammatory action, and coincident usually with the other appearances now described, has given rise to considerable diversity of opinion among pathologists regarding its origin.[3] It is almost invariably found in the neighbourhood of the pylorus rather than in that of the cardia, where only very slight traces of it can be discovered. The appearance described by Louis, of irregular prominences more or less rounded, of two or three lines in diameter, separated by sulci, and resembling

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presently to be noticed. In some of his cases the appearances described are those of the cicatrices of ulcers. (See Clin. Méd. ii. 153, 154.)

[1] This was noticed by Broussais (Lec. Phleg. Gastriques, 1823, p. 105), who gave it the term "coriaceous." I have frequently found the membrane almost as tough and resisting as leather.

[2] Rech. Anat. Path. 1826, p. 111, "Etat Mamelonné."

[3] See Hodgkin, Morbid Anatomy of Mucous Membrane, ii. 280.

the granulations upon wounds, when found in a healthy stomach most commonly results from the contraction of the muscular layer shown by Brücke[1] to exist around the basis of the secreting glands.[2] A very similar appearance may, however, be produced by hypertrophy and distension of the gland tubes with the products of secretion,[3] forming, together with the thickened interstitial tissue,[4] small granulations,[5] which are often rendered increasedly apparent by the atrophy of adjacent structures, and which thus present a counterpart in the stomach to the conditions observed in the granular kidney, and in cirrhosis of the liver.

Coincidently with the above, other changes take place in the secretory structures which must necessarily tend greatly to impair their functional activity. They may be briefly summed up as consisting in fatty degeneration of the glandular epithelium, associated with thickening of the membrana limitans, and finally tending to changes in the shape, or atrophy of the glands.

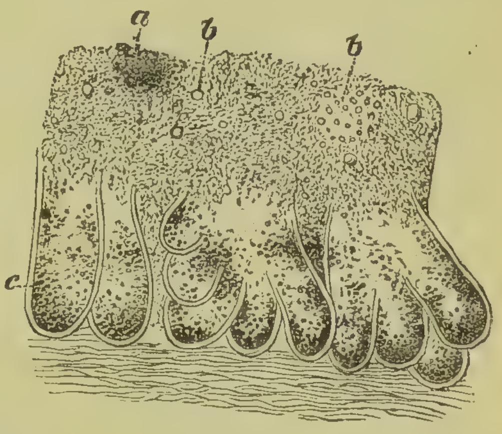

FIG. 5.—Tubular glands in advanced stage of chronic catarrh: tubes having a thickened membrana limitans (*c*) are filled with oil-globules. (*a*) Mass of pigment in tissue. (*b*, *b*) Free oil-globules.

Fatty degeneration of the glands generally occurs in the stomach, as in other glandular organs, in scattered groups, of one or two lines in diameter, giving the appearance of small, dead white spots imbedded in the mucous membrane. When microscopically examined, the epithelium of the tubes is found either fattily degenerated or the cells have entirely disappeared, and the contents of the tubules consist of nothing but free fat granules. The tubes are often irregularly narrowed and puckered, and thickening of the membrana limitans may not unfrequently be observed around their bases;[6] and these changes lead finally to the obstruction of the tubes at some part of their length, and to the formation of cysts, from the distension of the portion below the obstruction with the products of secretion.[1]

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[1] Bericht der Wiener Akad. 1851.

[2] If sections are made of membranes in this state, after hardening in chromic acid, it will be seen that the depressions consist of a group of from ten to twenty glands dragged down, as it were, below the surface of the others, but perfectly healthy in every other respect, and with no sign of atrophy of the glands, or of alteration of the surface.

[3] Andral, Clin. Méd. ii. 76.

[4] It is, I think, possible that the mere thickening of the interstitial tissue, especially when this is induced by a rapid increase of lymphatic growth, may in some instances alone suffice to induce this appearance.

[5] Polypoid growths of various sizes may form, especially in the pyloric region, which are due to the same cause (Rokitanski, Path. Anat. 1861, iii. 154, 155). They are not necessarily associated, however, with inflammation, though they are often very marked around cancers of the stomach. See also Andral, Prec. Path. Anat. ii. 50, 53; Clin. Méd. ii. 60. A complete monograph on this subject has been published by Ebstein, Reichert and Du Bois Reymond's Archiv, 1864.

[6] This, which is an inflammatory change, met with also occasionally in the acuter forms, requires to be distinguished from a fallacious appearance of the same kind which sometimes follows the addition of liq. potassæ or liq. sodæ to a section of a healthy mucous membrane. The pathological change may be recognised without reagents, or in glycerine, which does not produce this effect.

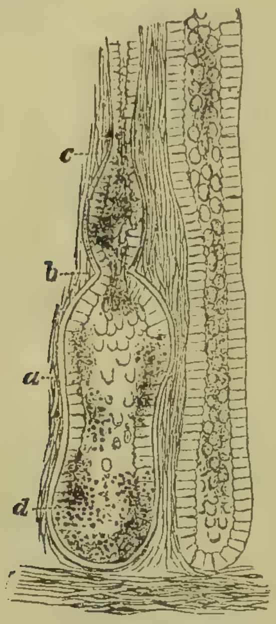

FIG. 6.—Commencing formation of cyst by constrictions of tubular glands at (*b*) and (*c*) : (*a*) thickened membrana limitans ; (*d*) fatty degeneration of contents of tube.

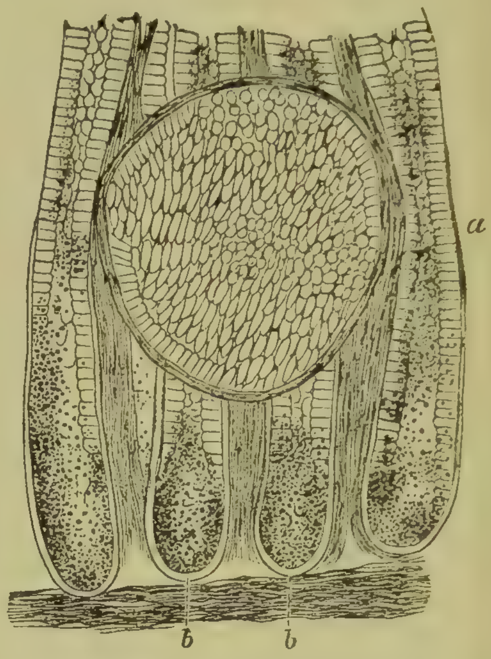

FIG. 7.—Cyst of stomach filled with columnar epithelium : (*a*) adjacent tubes, the contents of which are undergoing fatty degeneration ; (*b*, *b*) thickened membrana limitans.

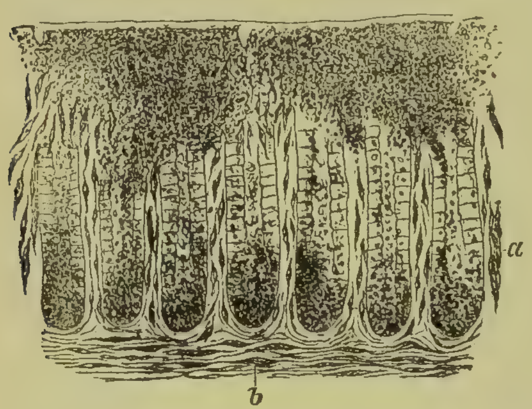

FIG. 8.—Fatty degeneration of mucous membrane : (*a*) fatty degeneration of intestinal tissue ; (*b*) fatty degeneration of corium.

Spots of fatty degeneration are also found, affecting both the gland-tubes and interstitial tissue, which sometimes extend through the whole thickness of the membrane, and are in some cases attended with a similar degeneration both of the capillaries and of the smaller arteries leading to the spot affected. These changes sometimes lead to a breaking down of the tissue, which resembles that seen in the superficial fatty degeneration of the lining membrane of the aorta, to which Prof. Virchow has applied the name of "fatty usur."[2]

Cases of chronic gastro-intestinal catarrh are sometimes found associated with the lardaceous, waxy, or amyloid degeneration; which, however, usually only occurs in these viscera when other tissues of the body, the liver, spleen, kidneys, and mesenteric glands, are largely affected with the same disorder. The extent to which the degeneration proceeds differs in individual instances, as also do the signs of the accompanying catarrhal action. During life, diarrhœa is often present, especially when leucocythæmia or albuminuria have co-existed;[1] in other instances I have observed this change associated with absolute anorexia, and in one case with great irritability of the stomach and vomiting. I have also found the disorder associated with much hyperæmia of the stomach, together with thickening and induration of the mucous membrane, and with fatty degeneration of the epithelium in parts where the specific lardaceous changes were but little apparent. These present the well-known reaction of a brownish-red colour with iodine, extending to variable depths of thickness in the mucous membrane, which is also usually simultaneously found in the villi of the intestines. In the stomach, in some instances, all traces of the epithelial cells are destroyed, and the contents of the tubes are converted into the refracting, homogeneous, irregular masses, into which the histological elements of the tissue are always changed in cases of this disease.

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[1] Further details on the structure and mode of origin of these cysts will be found in another work by the author, "Diagnosis and Treatment of Dyspepsia," and also in a paper by him, Med.-Chir. Trans. vol. xli. ; also in Dr. H. Jones's work, p. 115.

[2] Cellular Pathology, Chance's Translation, p. 340.

The exact relationship borne by this degeneration to the catarrhal conditions has not as yet been fully elucidated; it is probable that the two disorders may proceed *pari passu;* and such a combination is most prejudicial to the digestive process, as is seen from the rapid and marked emaciation which is so common an attendant on the lardaceous disease.

THE DIAGNOSIS of chronic catarrh of the stomach presents many difficulties, and in some cases the recognition of its etiological relationships is necessary for its successful treatment. Among these may be especially mentioned the forms when vomiting occurs from alcoholic excesses, from albuminuria, and from congestion through cirrhosis of the liver, to which, however, no further allusion appears necessary than the descriptions already given.

The chief points of distinction from atonic dyspepsia have been already alluded to.

The distinction between chronic catarrh and disturbance of the functions of the stomach from nervous derangement may be difficult in some cases of hypochondriasis, and also occasionally when vomiting forms a prominent symptom of the former class.

With respect to hypochondriacal affections, the diagnosis is often obscure, particularly as both forms are not unfrequently simultaneously present. Pain and severe uneasiness are more constantly complained of in hypochondriasis. Pyrexia, thirst, acidity, flatulence, or other disturbances of digestion, especially when associated with impairment of nutrition, are more distinctive of catarrh. The tongue also affords, in some cases, a reliable clue to the nature of the disease. The character of vomiting in catarrh is less easily mistaken for that of nervous origin. In the former it is rare and only occasional, and mucus is an almost constant product. In neurotic conditions the vomiting is never constant. It then occurs soon after food: it is rarely associated with cachexia, or with the signs of disordered digestion just indicated. The existence of an hysterical diathesis, or of some of the other distinguishing features of the neuroses, is usually also a sufficient guide to an accurate diagnosis.

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[1] Bennett, Princ. and Pract. Med. 532. Löschner und Lambl. aus dem Franz Joseph Kinder Spital, Beobachtungen und Erfahrungen, 1860, p. 341 et seq.

The diagnosis from ulcer and cancer may be doubtful in those cases in which vomiting is frequent, and especially when hæmatemesis occurs. Hæmatemesis in chronic catarrh is, however, almost invariably associated with congestion, and the diagnosis of its origin must depend mainly on the absence of severe pain, aggravated by food, and on the discovery of causes of obstruction to the venous circulation in the liver, heart, or lungs. On the other hand, when chronic inflammatory action is complicated by neuralgic pain, we have other criteria in the absence of hæmorrhage or of the signs of a tumour.

Tenderness on pressure also is entirely absent, or exists only in a much slighter degree in catarrh than in cases of ulcer and cancer.

THE TREATMENT of chronic catarrh of the stomach requires considerable diversity, according to the varied etiological conditions under which the disease may occur.

In cases succeeding an acute attack, the sedative plan of treatment is that which is usually followed by the most favourable results. Of all single remedies bismuth is the one which ordinarily proves the most efficacious, and it may often be advantageously combined with magnesia, or, where there is much nervous irritability of the stomach, with morphia and hydrocyanic acid, in the manner before mentioned. The chief indications for its use in this state are, pain or uneasiness at the stomach after taking food, together with a sense of load at the epigastrium, followed by acidity, and combined with a red and irritable tongue, or with one furred in the centre and red at the tip and edges.

The value of opium will be further treated of in relation to some of the special forms of the disorder.

In other cases of longer standing, more direct astringents are serviceable. The most useful of this class are the nitrate or oxide of silver, the former of which should be given in the solid form in doses of a quarter of a grain to one grain combined with opium, the latter in doses of one grain to two grains; the oxide of zinc, in doses of two grains to three grains; alum in solution, in doses of two grains to five grains; tannin or decoction of oak bark; and matico.

Arsenic in minute doses has been recommended by some writers as a valuable remedy. I have tried it in some cases, but hitherto without success: and we have not as yet obtained any definite indications for those in which it is likely to prove suitable; while it certainly aggravates the affection in cases for which it is not adapted.

The mineral acids are very useful, when given with meals, as aids to digestion, particularly when the atonic condition is also present.

It is only in marked forms of irritation that their use is contra-indicated. Their utility is sometimes considerable in the dyspepsia of phthisis, as pointed out by Trousseau,[1] though in this form, when the irritability of the stomach is very marked, sedatives and alkalies often succeed better—or at least until the acuter degree of the affection has subsided. They are also often of peculiar advantage in cases of irritative dyspepsia associated with deposits of oxalate of lime in the urine, and they are occasionally of service when the urine is phosphatic; but their influence in relieving either of these conditions depends on their power of improving the digestion, rather than on any effect produced by them on the composition of the urine, on the reaction of which they have very little direct influence.

Antacids and absorbents, administered between the periods of food, are useful when flatulence or acidity are present. Where the former predominates, magnesia suspended in equal parts of infusion of rhubarb and aq. menth. pip. often gives relief. Where heartburn alone is present, a scruple to half a drachm of the bicarbonate of potassa or soda may be taken dissolved in half a tumbler of water; and this treatment is sometimes beneficial when there has been a feeling of load and uneasiness three or four hours after a meal, accompanied with great physical languor and intellectual depression: the same advantage may also be obtained by drinking a tumbler of Vichy water between the meals, and also on retiring to rest at night.[2] They are further useful when urates or uric acid appear in the urine, and particularly in the acid dyspepsia of gouty cases. Caution, however, is required in order that the habit of taking these substances does not become confirmed, as more permanent injury to the digestion may result from their prolonged use.

Pepsine is also often of value in this disorder. I have found it especially so in the dyspepsia of scrofulous children, when the tongue is red and irritable; but I have employed it also under other circumstances with great utility.

Purgatives are only to be employed with caution, and cases of chronic irritative dyspepsia are often aggravated by a persistence in their use. In children especially, their frequent employment, and particularly that of mercurial " alteratives," is very undesirable.[3] Nor are these remedies indicated by the occurrence of pale, yeasty stools, which proceed as often from imperfect gastric digestion as from disordered " liver."

In some cases of long-standing, irritative dyspepsia, arising chiefly from excess in eating, benefit often accrues from free purgation with mercurials, and in gouty patients of this class colchicum may sometimes be advantageously added. Podophyllin is also of use in these circumstances.

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[1] Traité de Thérap. i. 206. Clin. Méd. ii. 1862, p. 337.

[2] Whytt (Works, p. 664) recommended, for persons troubled with mucous vomiting, a tumblerful of lime water, to be drunk on an empty stomach in the morning.

[3] "And with reference to mercury, I would advise you to have your grey-powder bottles marked *Dangerous, especially in alterative doses.*" (Sir W. Jenner, Lectures on Rickets, Med. Times and Gazette, 1860, p. 446.)

Mercurial purgatives are also of use in cases of congestion of the stomach arising from disorder of the liver, even when this is so severe as to give rise to hæmatemesis or melæna.[1] They have also favourable effects in some cases of this class, when pain is present, associated with the vomiting of ropy mucus,[2] though in these the subsequent administration of astringents is necessary to complete a cure.

Cases occasionally occur when a severe and long-continued inflammatory condition of the stomach, which has resisted all other remedies and also a careful dietetic regimen, yields promptly to a mild mercurial course sufficient to touch the gums; after which, medicines that had previously been unavailing have proved beneficial.[3] The same method has been followed by the author with success in cases when there was a suspicion of a syphilitic origin of the disorder of the stomach.[4]

Habitual constipation may be relieved by two or three grains of the pill aloës dil. taken with food, which may sometimes be advantageously combined with the extract of nux vomica; or by two or three drachms of the decoct. aloës aquos. taken before rising in the morning. When piles exist, the action of the bowels may be procured by the use of the Püllna or Friedrichshaller waters, or by a few drachms of the potassio-tartrate of soda, taken before breakfast. Castor-oil also proves an excellent laxative in such cases; but the use of all these remedies should be avoided as far as possible, and the action of the bowels should be solicited by the daily use of enemata of cold water.

The employment of belladonna in doses of one-tenth to half a grain, as recommended by Trousseau,[5] also proves occasionally beneficial.

Many of the natural mineral waters have a very decided beneficial effect in restoring a healthy condition of the functions of the alimentary canal when suffering under chronic catarrhal affections. The most valuable in this respect in our own country are those of Harrowgate, Bath, and Leamington. The waters of Carlsbad and Marienbad are also valuable in these complaints—the former being most useful when there is much portal congestion, and the latter when the disorder depends more on simple irritation of the gastro-intestinal canal, and also in young persons who suffer from constipation.[6] The mineral waters of Kissingen, containing a large proportion of iron, are also of service when general atony or anæmia is combined with the irritative state of the mucous membrane. Those of Vichy are of great general utility as alkaline remedies, and are also specially applicable to dyspepsias of gouty origin; and though their efficacy is less explicable, the springs of Pouges, Plombières, and Bagnerres de Bigorre have obtained a high reputation.[1]

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[1] These are the cases in which purgatives prove efficacious in the relief of this symptom (Sir T. Watson, loc. cit. ii. 435); but great care is necessary in the diagnosis, as they aggravate those in which the hæmorrhage proceeds from ulcer or cancer. In some instances, however, the hæmorrhage from congestion may be sufficiently severe to threaten life, under which circumstances the ordinary means for checking the flow must be resorted to.

[2] Barlow, art. "Gastrodynia," Cyc. Pract. Med. iii.

[3] This plan, laid down by Dr. Hunt (Heartburn and Indigestion, p. 73 et seq.), has been illustrated by some cases of Dr. H. Jones.

[4] See also Trousseau, Traité de Thérap. i. 269; Andral, Clin. Méd. ii. p. 201 et seq.

[5] Clin. Méd. ii. p. 381.

[6] Oppolzer, Zeits. Gesellsch. Aertzte zu Wien. Canstatt, 1857, iii. 175.

The condition arising from the abuse of alcoholic stimulants requires certain modifications of the plans above indicated. Occasional mercurial purgatives prove most undoubtedly beneficial; but when there is much irritability of the stomach or vomiting of mucus, opium[2] has a special value. Its action may often be assisted by its combination with astringents, and particularly with the compound kino powder. Its beneficial effect appears to be of a twofold character: locally, it allays the irritation of the stomach, and checks the excessive mucous secretion; while, by tranquillizing the nervous system and procuring sleep, it restores the tone of the digestive organs, and frequently enables the patient to digest solid food. Opium is also of use in cases of subacute inflammatory action, combined with great nervous irritability, and where atony also exists; it may often be given with advantage combined with nitrate of silver, as before recommended.

The simple bitters may be used in these cases when the acuter symptoms have subsided. Strychnia, or nux vomica combined with the mineral acids, or the oxide of zinc, are also remedies which are specially applicable to the state of combined disturbance of the nervous system and of the digestive functions which these cases exhibit.

The disturbance of the digestive organs associated with albuminuria is often greatly relieved by free purgation with the compound jalap powder, as pointed out by Dr. Budd.[3] The vomiting is, according to the author's experience of the disease, more effectually checked by ice than by any other remedy. In these cases an animal diet often suits better than a vegetable, and large quantities of slightly underdone meat may sometimes be taken with considerable advantage. Creosote, as recommended by Rayer,[4] as a means of checking vomiting from this source, is sometimes also useful; but its efficacy is somewhat uncertain.

The irritative dyspepsia of phthisis offers greater difficulties to a complete cure than almost any other of the forms of this disorder. One essential point to be borne in mind with regard to it is the necessity of chiefly employing sedative remedies; and of these I have found none so efficacious as hydrocyanic acid, the value of which remedy is, I believe, more marked in this disorder than in almost any other form of dyspepsia. It may be most usefully combined with the carbonates of potassa or soda, and given, in combination with infusion of calumba, twice or three times daily, in the intervals of meals.

Bismuth may also be employed with advantage, especially when there is diarrhœa, under which circumstances its combination with opium or morphia is indicated.

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[1] Trousseau, Clin. Méd. ii. 379.

[2] Andral, Prec. Anat. Path. ii. 204; Budd, Dis. Stomach.

[3] Loc. cit. p. 248.

[4] Mal. des Reins, ii. 347.

The dyspepsia of scrofulous children—in whom the tongue is red and irritable, the complexion sallow, the spirits uneven, the general strength deficient, as shown by frequent complaints of lassitude, the appetite irregular and often voracious, but not unfrequently perverted, and the bowels irregular (the motions being sometimes loose and pultaceous, and at others scanty and confined, but generally pale and offensive); and who often complain much of thirst—or of those of relaxed and atonic constitutions, in whom the external signs of scrofula are not well marked, is another form which requires great care both in treatment and diet. This caution applies especially to the use of purgatives, and particularly to mercurials, which seldom fail to aggravate the condition.

The first and most essential point to be attended to in such cases is the regulation of the diet, both as to quality and quantity: *all* indigestible substances are to be carefully eschewed, and the amount of animal food is to be strictly limited until marked improvement in the digestion has taken place.[1] Great care is necessary also in ensuring the due mastication of food; and when children have acquired the habit of performing this imperfectly, they often require to be carefully watched during some weeks, until it has been overcome. Animal food may in such cases often be advantageously minced; but it must be remembered that the necessity of mastication is equally to be insisted on with all the food taken.

Mercurial purgatives are contra-indicated, and constipation is to be met by small doses of rhubarb and soda, or by castor or olive oil. A few stewed prunes may often be advantageously allowed with the meals; but other fruits, with the exception of strawberries, are, as a rule, to be avoided. The use of vegetables is only to be very cautiously permitted; and in severe cases they are to be forbidden.

As long as marked irritation persists, it is desirable to continue the use of sedatives, and of these bismuth is the most efficacious; but small doses of the carbonated alkalies, with one or two drops of dilute hydrocyanic acid combined with infusion of calumba, may be given.

The condition of atony which underlies these cases is best treated by pepsine and iron; the latter should be given in the neutral form or combined with alkalies; and when the stomach permits of its use, the administration of cod-liver oil is often markedly beneficial. General hygienic measures are also carefully to be observed, and particularly the use of warm clothing;[1] sufficient exercise in the open air, and good ventilation of the day and sleeping apartments. Cold or sea bathing must be regulated by the vigour of the reaction of the skin. Sponging with salt and water is sometimes advisable.

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[1] In carrying out this system the physician will often have to encounter no little difficulty from the friends of his patient, who, seeing the loss of flesh and strength, not unfrequently endeavour to remedy the weakness by increased food, wine, and tonics—a plan which unfailingly tends to aggravate the symptoms, whilst an opposite procedure, during a limited period, is often productive of the best results. I have known the most obstinate irritative dyspepsias of this nature cured under a system of diet from which, during nine weeks, animal food has been almost entirely excluded, and the patient restricted to the use of light farinaceous puddings and bread and milk.

The warm bath, on the other hand, is frequently beneficial when the stomach is irritable, and particularly so in children of gouty or rheumatic parents.

A very important point to be recollected in these cases is that they are liable to frequent relapses, and that under such circumstances a return for a few days to a restricted diet and a sedative treatment becomes absolutely necessary.

The weakened state of the digestion, which often remains long after signs of irritation have disappeared, requires the cautions respecting the treatment of such cases given in the chapter on Atonic Dyspepsia. The amount of food taken should be regulated by the digestive power of the stomach.[2] Stimulants should only be taken in great moderation, and of these the drier varieties of sherry, or claret, or Chablis, should be preferred.

The advantages of moderate exercise, and of change of air and scene, cannot be too strongly insisted on, and are often productive of the happiest results. One very important point to be attended to is that patients should always wear flannel, and be sufficiently clothed to protect them against the effects of change of temperature, to which they are peculiarly liable; and the general regimen of atonic dyspepsia should be most carefully observed.

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[1] The exposure of young children of delicate constitutions to cold, by imperfect clothing, in the manner so commonly practised, cannot be too strongly reprobated.

[2] It is very difficult to prevent patients in this condition from eating more than their stomachs can digest, under the erroneous idea of thereby regaining health and strength: it is not uncommon to find feeble subjects of irritative dyspepsia using very little exercise, and taking meat three times daily, together with a considerable quantity of stimulant, and with beef-tea once or twice in the intervals, who improve at once and rapidly on submitting to a more restricted diet. The just medium in these cases is at all times one difficult to attain, but the effects of a certain degree of abstinence are often most beneficial, and it is rarely that it is carried too far, at least in comparison with excesses in the opposite direction.

VI.—Chronic Ulcer of the Stomach and Duodenum.

Synonyms.—Perforating Ulcer (Rokitanski); Simple Ulcer (Cruveilhier); Round Ulcer; Corrosive Ulcer (Müller); Ulcer of Stomach (Brinton).

Definition.—A disease characterised during life by pain in the stomach, and usually associated with vomiting, hæmorrhage, and disturbances of the digestion, and terminating either in cure, or in death by hæmorrhage, perforation, or marasmus. Its essential anatomical character consists in a circumscribed loss of substance of the coats of the stomach or intestine, extending for a variable depth through their tissues, which is sometimes associated with inflammatory thickening of its margins, but is not attended by any other morbid growth.

History.—It is probable that a large number of the cases described by the earlier writers under the titles of Cardialgia, Gastrodynia, Hæmatemesis, and Melæna were really referable to this disorder. Ulcerations of the stomach were indeed recognised by Celsus,[1] and mentioned in several places by Morgagni,[2] but their effects were confounded by other writers with rupture, either spontaneous or from violence; or they were described among the appearances produced by chronic gastritis or duodenitis (Andral, Broussais, Abercrombie). The first authors who distinctly traced the connexion of the special symptoms characterising this disease with a definite anatomical alteration were Cruveilhier and Rokitanski, who gave complete descriptions of its leading features, which have since sufficed for the basis both of diagnosis and treatment. Subsequent additions have been made to our knowledge of its pathology and etiology by Prof. Virchow and by Dr. Brinton, the latter of whom has given statistics based on a wider comparison of the published cases than had previously been attempted; and the same course has been followed by other writers mentioned below.[3]

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[1] De Med. liv. cap. 5.

[2] Epist. lxv. 3, xxix. 14, 20, lxix. 3 (Ulcerations of Stomach and Duodenum from Arsenic).

[3] The principal literature of this subject is to be found in the following works:— Baillie, Morbid Anatomy. Hope, Morbid Anatomy. Cruveilhier, Revue Médicale, 1838; Archives Générales de Méd. 1856; and Path. Anat. du Corps Humain, Liv. xxx., xx., xxvii. Rokitanski, Med. Jahrbücher des Oesterreichen Staates, 1839, and Path. Anat. Albers Beobachtungen. Reports by Jaksch, Willigk, Dittrich, and Duchek, of clinical and post-mortem observations in the Prager Viertel-Jahreschrift, vols. iii., vii., viii., ix., xii., xiii., xiv., xxxviii., xliv., l., li. Langston Parker, Stomach and its Mor- bid States, 1838. Williamson, Dublin Journal, 1841. Crisp, On Perforation of Stomach, Lancet, Aug. 5, 1843. Osborne, Dublin Journal, 1845. Virchow, Archiv Path. Anat. v. 275 et seq. Chambers, Lond. Journ. Med. 1852. Handfield Jones, Med.-Chir. Trans. 1854, and Path. and Clin. Obs. respecting Morbid Conditions of the Stomach, 1855. Budd, Lectures on Diseases of the Stomach, 1855. Brinton, British and Foreign Med.-

ETIOLOGY.—*Frequency of occurrence.* The largest data on which an estimate can be formed of the proportionate number of cases in which this disease occurs are to be drawn from the returns of Jaksch, Dittrich, and Willigk, from the hospital at Prague, who in a total of 10,203 bodies examined, found 126 open ulcers and 224 cicatrices in the stomach and duodenum,[1] representing a frequency of 3·4 per cent., a result which corresponds tolerably closely with those of Dr. King Chambers and Dr. Brinton.

The returns of Dahlerup,[2] from the hospital of Copenhagen, present a marked contrast to those just quoted: in 200 bodies he found 20 open ulcers and 6 cicatrices, giving a percentage of 13 to the total number of deaths. It is doubtful whether the marked discrepancy between this observation and that of other authorities is explicable by the small number of bodies on which it is based, or by special circumstances affecting the population.

Age.—Dr. Brinton's return of 226 cases of ulcers and cicatrices, collectively, in which the age is mentioned, shows a gradually increasing frequency of occurrence with advancing years:—

Between ages of	0 to 10	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60	60 to 70	70 to 80	80 to 90
No. of Ulcers .	2	18	45	39	38	32	32	15	5
	65			77		84			

and allowing for the number of all persons living at these ages, the apparent preponderance of the disease in the later periods of life is very considerable.[3]

Chir. Rev. xvii. 1856; Ulcer of Stomach, 1857. Habershon, Obs. on Alimentary Canal, 1857. Luton, Rec. des Travaux de la Soc. Méd. d'Obs. 1858, vol. i. Müller, Das corrosive Geschwür in Magan und Darmkanal. The two last-named authors give a full historical account of the literature of this disease. A synopsis of the literature and valuable critical observations on the etiology of the disorder are also to be found in an article by Miquel in the Hannoverische Zeitsch. für praktische Heilkunde, 1864. For description of duodenal ulcers see Budd, loc. cit.; Mayer, Krankheiten des Zwölffinger, Darmst. 1844; and for wider statistical observations see Trier, Ulcus Corrosivum Duodeni, reprinted from the "Ugeskrift für Læger," Copenhagen, of which an abstract is given in the British and Foreign Med.-Chir. Rev. Jan. 1864, and in the Prager Viertel-Jahresch. vol. lxxxv. Also Krauss, "Das perforirende Geschwür in Duodenum," 1865. For the origin of this latter disorder, in connexion with burns of the skin, see Curling, Med.-Chir. Trans. xxv.; Erichsen, Lond. Med. Gaz. 1843. See also Abercrombie, Diseases of Abdominal Viscera. Bennett's Clinical Medicine. Henoch, Klinik der Unterleibs-Krankheiten, and Bamberger, Krank. des chylopoietischen System, Virchow's Handbuch der Spec. Path. Thérap. vol. vi. For some of the references to the less accessible cases, especially in the German medical journals, the author is especially indebted to the works of Müller, Miquel, and Krauss.

[1] Dittrich's cases, I believe, include duodenal ulcers. He distinctly mentions one such, but in most of his returns the site of the ulcers is not mentioned.

[2] De Ulcere Ventriculi Perforante; Havniæ, 1841. Canstatt's and Eisenmann's Journal, 1842. The original of this work appears to have been inaccessible to most of the writers who have quoted it, as it has also been to myself.

[3] There are, however, two fallacies in such an estimate: for in the first place the appearance of the cicatrix gives no information at what age the ulcer from which it may have resulted has occurred; secondly, the duration of life, in many cases even of open ulcer, is often very considerable, and therefore the discovery of either lesion at an advanced age affords no certain criterion of the date of its origin. That the ulcers may,

The disease is rarely met with in the earlier periods of life; but a doubtful case is recorded in a newly-born infant,[1] another instance is mentioned by Barrière,[2] in which an ulcer was formed in the duodenum in a child of six years old, Dr. Budd[3] has met with it in a girl ætat. 14½, and Dr. Brinton[4] and Dr. Buzzard[5] have found it at the ages of 8 and 9.

Other returns, though based on smaller numbers, show that there is a preponderating liability to the disease between the ages of 15 and 30.

Thus a comparison of the tables of 108 fatal and open ulcers given by Willigk and Miquel affords the following results:—

Age.	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60	60 to 70	70 to 80	Total.
Ulcers—Males . . .	2	9	6	8	5	2	1	33
,, Females . .	13	21	9	15	6	8	3	75
Total	15	30	15	23	11	10	4	108

And of 21 not fatal, by Duchek, the ages in which they occurred were:—

Age	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60
Ulcers	3	6	7	4	1

Dr. Crisp, of 51 cases of perforation, of which 39 occurred in females and 12 in males, gives as the ages of the former:—

Age	15 to 30	20 to 25	25 to 30	30 to 60
Ulcers	21	10	5	3

Sex.—The disease appears, from the returns of numerous observers, to be between twice and three times as frequent in the female as in the male sex.

The sex of the patient appears also to exercise an influence not only on the age at which the ulcer appears, but also on that at which one of its most fatal accidents, viz. perforation, occurs, and probably in some degree on the special liability to this event. Thus by comparison of the tables of open and fatal ulcers given by Willigk[6] and Miquel it will be seen that, in the male sex, only one-third occurred before

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however, frequently commence at an advanced age, is seen by a case of Cruveilhier's, Path. Anat., Liv. xx., and also by several recorded by Dr. Brinton (Ulcer of Stomach, Appendix).

[1] Busch, Hufeland's Journal, 1836.

[2] Malad. de l'Enfance, ii. 1, quoted by Henoch, ii. 130.

[3] Loc. cit. 115.

[4] Loc. cit. 33.

[5] Path. Soc. Trans. xii. 84. All the three last-named cases had perforated.

[6] In Willigk's cases the whole number of females examined exceeded that of the males, being 3,440 of the former to 2,766 of the latter.

the age of 30 (11 to 22), while of the females the proportion of cases occurring within this period to those observed at all other ages was as 34 to 41. The greater liability to perforation at early ages in the female sex is apparent from Crisp's tables, and also from a large number collected by Dr. Brinton. These show that nearly three-fourths of the instances of this event in the female sex occurred before the age of 35, while in the male sex the cases of perforation are nearly equally divided among all ages, though somewhat diminishing in frequency after that of 50 had been attained.

The absolute liability to perforation in the two sexes is a matter of some uncertainty. Dr. Brinton estimated it as about corresponding to the absolute proportionate frequency of ulcerations observed,[1] but other authorities believe that there is a greater relative frequency among females.[2]

Of the influence of race and climate but little is known. But few positive facts are known regarding any influence exerted by occupation, though Dr. Copland asserts that, when occurring in the female sex, it is most common in those engaged in needlework or in domestic service.[3] There is a general impression that the disease is most common among the poorer classes, but no certain data exist for this opinion.

Among other occasional causes which have been mentioned, are moral emotions,[4] bad or insufficient food, excessive indulgence in spirituous drinks,[5] and exposure to extreme cold.[6] *Corrosive poisons*, and particularly the mineral acids, may also give rise to perforating ulcer:[7] their *modus operandi* will be easily understood when the pathology of the disease is considered.

There are certain *diseases* to which a direct causative influence has been ascribed in the production of the complaint, and which deserve a passing notice.

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[1] Dr. Brinton's estimate of the relative frequency of the ulcer in the two sexes gives a smaller preponderance to the female sex than do the numbers collected by other observers.

[2] In 221 cases of ulcer by Willigk there were six perforations, all in the female sex; of sixty-seven cases of perforation by Miquel, fifty-one were females; while Dr. Crisp's tables, before quoted, give a proportion of thirty-nine females to twelve males.

[3] Med. Dict. iii. pt. 2, p. 919.

[4] See a case by Cruveilhier, Path. Anat., Liv. xx. Osborne also says that he has known the symptoms of ulceration to date from this cause; Dub. Med. Journ. xxxvii. p. 357.

[5] Leudet, Congrès Méd. de France, 1868, 104. In twenty-six cases of drunkards, Leudet found eight cases of ulcer, open or cicatrized. He calculates that one-third of the whole number of ulcers found by him were associated with habits of intoxication. See also Klob, Bericht über die Wiener Sammlung (Canstatt's Jahresb. 1850, quoted by Leudet); also Huss, Alcoholismus Chronicus, p. 5. Leudet further gives cases where hæmatemesis followed the drinking of large quantities of alcohol.

[6] A case by Förster, Wurzb. Med. Zeitsch. 1864, ii. 164. The etiology of this case is doubtful. The patient had been exposed to great cold, and drank a large quantity of raw spirits. There was perforation of the posterior wall of the duodenum with an abscess in the retro-peritoneal tissue. Förster thought it possible that the spirits might have produced an excessive secretion of gastric juice leading to the perforation.

[7] See a case of perforating ulcer of the stomach caused by swallowing hydrochloric acid, related by the author, Path. Soc. Trans. xix. 239.

Amenorrhœa is perhaps the one which holds the most important rank among these, but precise information is wanting regarding the exact relation of the two disorders. It appears to be pretty certainly established that, though menstruation is sometimes regularly maintained after ulceration, especially in middle-aged females (Brinton), yet that disturbances in this function accompany ulcers of the stomach with a greater proportionate frequency than is met with in almost any other class of disorders, except perhaps tuberculosis. We possess, however, no certain information as to the number of instances in which suppression or arrest of the menstrual flow has preceded the disease of the stomach. Cases are indeed recorded in which suppression of the menses, through cold, in previously healthy females has been immediately followed by symptoms of ulceration; and in some, even after the re-establishment of the menstrual function, the symptoms of the disease of stomach recurred with each return of the uterine discharge.[1] The age at which the disease is found with such frequency in the female sex appears to point in a very distinct manner to the influence of puberty on its occurrence, and Miquel[2] states that he has observed a similar though less marked liability at the climacteric period between the ages of 40 and 50. These facts, together with the evidence afforded by Crisp[3] of the association of amenorrhœa with the liability to perforation, would tend to show that the connexion between the two conditions is something more than accidental, and that, as will be further considered when the pathology of the disorder is discussed, these disturbances, when preceding the appearance of the ulcer, may be reasonably supposed to have a direct influence on its origin.

The influence exerted by the states of anæmia or chlorosis on the occurrence of ulceration is less evident. They have been very frequently observed in cases of perforation,[4] which, under these circumstances, may in part be due to the absence of sufficient vitality to allow of the projective thickening of the edges and base of the ulcer, by which this accident is commonly prevented. It is stated, however, by Miquel, that a sound condition of health has often in the female sex immediately preceded the ulceration.

Tuberculosis is met with in a great number of cases, but, as shown by Dr. Brinton, "the percentage of tubercle in cases of gastric ulcer does not seem to exceed its average in all persons indifferently;" nor do other diseases of the lung appear to exert any direct influence on its production.

The coincidence of the ulcer with the puerperal state mentioned by Chaussier has been brought into prominence by Jaksch's returns, who found that, of 91 females, it occurred in 10 during the period of child-bed. Rokitanski was of opinion that the occurrence of intermittents predisposed to the disorder, and Engel[1] says that in 10 per cent. of his cases the patients were syphilitic. Krauss says that the disease has commenced after suppression of hæmorrhoidal discharges; Miquel, after the healing of old ulcers on other parts of the body.[2] The association of the disorder with diseases of the liver will be alluded to in the history of its pathology.

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[1] See Miquel, loc. cit. 145.

[2] Loc. cit.

[3] Dr. Crisp found that, in thirty-nine cases of perforation, the state of the menstruation was not mentioned in twenty-five. In the remaining fourteen, it was present and regular, in one; irregular, or suppressed, or had never appeared, in thirteen.

[4] Especially by Crisp.

Of the other diseases with which it is most commonly found associated may be mentioned those of the heart, though by no means exhibiting a great frequency of occurrence in proportion to the whole number of cases dying of these disorders.

Another affection which appears to act as a direct cause of these ulcers, but the effects of which are remarkable as being almost wholly confined to the duodenum, are burns of the skin. Their influence in this respect was pointed out by Cumin,[3] Dupuytren,[4] Cooper,[5] and Long,[6] and was more fully brought forward by a collection of cases published by Mr. Curling,[7] and in a memoir by Mr. Erichsen.[8] Mr. Holmes[9] has recently given further illustrations of their comparative frequency; in 125 cases of severe burns collected by him, the duodenum was ulcerated in 16, and other portions of the intestines in 2 others. The earliest period at which the ulceration has been discovered has been on the 4th, 5th, or 6th day. The age of the patient exercises no influence on the result, nor does apparently the situation of the external injury.

MORBID ANATOMY.[10]—It has been already stated, in the definition above given, that the essential anatomical alteration of this disease is an ulcer or ulcers of the coats of the stomach.

The term as thus used is not, however, applied to all forms of ulceration of the mucous membrane, and it does not embrace either the superficial or the follicular erosions, which have been previously described as found in catarrhal inflammations of the organ. Nor, in the author's opinion, should the superficial hæmorrhagic erosions be included in the definition of this disease, though its relation to the latter form will be again alluded to.

As distinguished from the above forms, the chronic or perforating ulcer of the stomach is one which extends for a greater depth in the tissues, sometimes passing only through the mucous membrane, at others penetrating the muscular tissue, and even the peritoneum.

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[1] Prager Viertel-Jahresch. vol. xl. p. 7. As, however, he does not give the whole numbers on which this calculation was founded, its relative value can scarcely be estimated. The evidence of disturbance of the stomach from a syphilitic taint does not show conclusively that the cause is due to ulceration. See Andral, Clin. Méd. ii. 201 et seq., and Höring, Schmidt's Jahrbücher, 88.

[2] Two cases, loc. cit. 143. See also a case quoted by Cruveilheir, Liv. xx., where the disease in the stomach commenced in a patient æt. 62, after the healing of an ulcer in the skin.

[3] Edin. Med. Surg. Journ. 1823.

[4] Leç. Orales, p. 521.

[5] Lond. Med. Gaz. xxvii.

[6] Lond. Med. Gaz. Feb. 1840.

[7] Med.-Chir. Trans. 1842, vol. xxv.

[8] Lond. Med. Gaz. 1843, 789, 790.

[9] Syst. of Surgery, vol. i. p. 733 et seq.

[10] The clinical history of this disorder is in many points so fully elucidated by the pathological conditions observed in the stomach, that a departure from the usual order followed in this work appears in this instance desirable.

(*a*) *Character and Appearance of Ulcer.*—It is found in two conditions, which, as they are supposed to represent either different stages or variations in its mode of progress, it seems not unimportant to distinguish from one another.

The first of these, which is believed to be that in which the ulcer is found soon after its origin, presents a sharply defined loss of substance of variable depth in the coats of the stomach: it may then, when affecting only the mucous membrane, have a flattened appearance, with clean-cut edges, looking as if they had been punched out of the tissue; when, however, the destruction has extended more deeply, it generally presents, even at this period, an appearance somewhat resembling that of a funnel whose apex is directed towards the peritoneum.[1] The edges and floor in this stage are smooth and even, the former seldom presenting any swelling or elevation above the surrounding tissue; the base, however, often shows a softened, puffy look, and minute sloughs may sometimes be found upon it. In some cases, however, the ulcer has been found with a mass of blackened blood adhering to its base,[2] or with an infiltrated extravasation of blood into its margins, or surrounded by a zone of injection in which petechial extravasations have occurred,—appearances which, as will be seen, are of considerable interest in relation to the pathology of this disease, and to the experiments conducted with a view to its artificial production in animals.

In this stage the ulcer frequently perforates; but the morbid anatomy of this result of the disorder will be separately described.

When the ulcer has existed for a longer period, its walls and floor undergo an inflammatory thickening, through which its funnel-like shape becomes more distinct, and it acquires a stratified and crater-like appearance, which is less distinct in its earlier stages. This appearance is due to the different coats being involved in areas which progressively diminish from within outwards, the mucous and submucous tissues being destroyed over a wider surface than the muscular, which thus forms the first step, and through the opening in which the thickened peritoneal and sub-peritoneal tissues are seen penetrated in a smaller extent by the progress of this disease.

The whole tissue of the wall is thickened around the ulcer, and the different coats become blended together, sometimes for a considerable distance around its margin. The new tissue by which this is effected consists of an amorphous or finely granular intercellular substance, in which are imbedded nuclei more or less thickly scattered. At a later period it is developed into an imperfectly fibrillated tissue. Occasionally, but not commonly, granulations may be seen on the sides and base of the ulcer.

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[1] This funnel-shaped opening, as has been observed by Virchow, does not always extend vertically into the tissue in its whole circumference, but one side may be vertical and the other sloping.

[2] Frerichs, Dis. of Liver, Syd. Soc. Trans. by Murchison, Case I. vol. i. p. 136. Bennett, Clinical Medicine, 789. Habershon, Obs. Aliment. Canal, 1862, p. 98. Also a case quoted by Mr. Curling from Long, Med.-Chir. Trans. xxv. 269. Also Handfield Jones, loc. cit. 128.

The tubular glands in the immediate neighbourhood are sometimes destroyed by this growth, or villous or polypoid vegetations may be developed on the mucous surface surrounding the ulcer; but in the majority of cases these appearances are not observed, and the ulcer exists only as a conical-shaped perforation of the coats of the stomach, surrounded by a thickened and indurated cicatricial margin, and resting on a base having the same characters. In rarer instances the ulcer has been found associated with suppuration in the coats of the stomach, leading to secondary thrombosis and suppuration in the portal vein.[1]

In some cases the retraction of the deeper layers may give the mucous membrane an excavated appearance, and this may proceed to the extent of causing the edges of the mucous membrane to meet in the centre (Krauss); but, ordinarily, when cicatrization ensues, it proceeds by granulations from the base, and the previous site of the ulcer is marked by a cicatrix of variable size, uncovered by mucous membrane, and surrounded by stellate, radiating lines extending into the surrounding tissue.

(*b*) *The size* of these ulcers varies from that of a fourpenny-piece to a diameter of five or six inches.[2] The larger diameters mentioned have been almost entirely met with in cases where there was reason to believe that the disease had been of long standing; and those of more recent formation, or which have run an acute course, have seldom exceeded that of a shilling or half-a-crown. Perforating ulcers have been artificially produced[3] not larger than a pin's head in diameter.

(*c*) *The shape* of the ulcer is usually round or ovoid. Coalescence of one or more may give, however, various irregularities of outline; and instances are recorded in which they have been found surrounding the whole circumference of the pylorus.

(*d*) *The number* met with in any single subject varies.

Dr. Brinton says that, out of 536 cases, two or more were present in 113, or in 21 per cent.[4] Instances, however, are mentioned in which three, four, five, or even more open ulcers have been found in the same stomach; and Krauss says that, in the cases of duodenal ulcers observed, a plurality has been found in one-third. The simultaneous occurrence of ulcers in the duodenum and in the stomach is also, comparatively speaking, a frequent event.

(*e*) *The seat* of the ulcer is much more commonly in the stomach than in any other portion of the intestinal canal. It is very seldom met with in the œsophagus,[1] and is much less frequent in the duodenum[2] than in the stomach; but instances are recorded in which similar ulcers have been discovered in lower portions of the intestines.[3]

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[1] Dr. Bristowe, Path. Soc. Trans. ix. 275.

[2] A case is given by Law, Dub. Hosp. Gaz. ii. 51, of an ulcer measuring 6 in. by 3 in., quoted by Lees (Diseases of Stomach).

[3] Müller, loc. cit. 273.

[4] He does not, however, state whether these include cases where recent ulcers and cicatrices of older ones were present together,—a very common condition.

The seat of the ulcers in the stomach exercises an important influence on the progress of the disorder, and therefore deserves especial mention. The largest statistics on this subject are given by Dr. Brinton, who, in 220 cases in which the site was recorded, found that eighty-six were on the posterior surface; fifty-six on the smaller curvature; thirty-two on the pylorus; thirteen on the anterior and posterior surfaces, which frequently existed together, and were often opposite to one another; ten on the anterior surface only; five on the greater curvature; and four in the cardiac pouch. Rokitanski's remark, that the ulcers are more commonly seated in the immediate neighbourhood of the curvatures rather than directly upon them, has been fully confirmed by more extended observation.

The majority of ulcers in the duodenum are situated in the upper horizontal portion.[4]

(*f*) *The progress* of the ulcer, when once formed, tends either to cicatrization or to extension through the coats of the organ; in the latter case leading either to perforation of the stomach or to the invasion of neighbouring organs through adhesions previously contracted.

(*g*) That *cicatrization* is by far the most common result is shown by the returns before quoted, where it is seen that cicatrices are found nearly twice as frequently as open ulcers.[5]

The process by which cicatrization is accomplished has been already described, but its final results differ considerably, according to the depth and superficial extent of the ulcer, the adhesions which it has contracted to neighbouring organs, and the amount of thickening which has taken place around its base and margin.

In many cases the process is not complete, and there is a proclivity to a return of the ulcerative action in the cicatricial tissue.

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[1] A case of this kind is given by Mr. Flower, Med.-Chir. Trans. xxxvi.

[2] Willigk found, for 225 cases in which the stomach was affected, only 6 cases of ulcer in the duodenum; and Trier, on the other hand, gives, as the relative proportional frequency in these parts, 28 cases of duodenal ulcer, as contrasted with 261 where it was found in the stomach. It is not, however, unlikely that cicatrices in the duodenum may be frequently overlooked: so that no absolute reliance can be placed on these numbers.

[3] Albers, Die Darmgeschwüre, p. 474 et seq. Lebert and Clauss, Ueber spontan. Darm-Perforationem, Diss. Inaug., Zurich, 1856,—a case where a perforating ulcer existed in the colon simultaneously with another in the stomach (quoted by Krauss); Lebert, Handb. der Prakt. Med. ii. 369. Dr. Dickinson, Trans. Path. Soc. 1867, has recorded a case of ulceration in the colon. I have seen two cases where intermitting hæmorrhage from the colon rendered the diagnosis of ulcers in this part of the bowel extremely probable. Both were in females. Cases, where large alterations in the intestines resulted from embolism, are given by Panum, Virch. Archiv, xxv. See also on this subject Mr. Holmes's article, before quoted.

[4] Of 47 cases collected by Krauss, only 2 were situated in the lower horizontal part.

[5] Dr. Brinton, from a smaller number of cases, deduced that open ulcers and cicatrices are met with in about equal frequency. The result of the larger returns points to an interesting fact as regards the curability of the disease, which will be the subject of further remark.

When the loss of substance has not penetrated deeply, or when cure has followed rapidly on the destruction,[1] the site of the injury is only marked by a white spot in the mucous membrane, attended with little or no puckering or contraction; but in other cases a similar appearance, with radiating, stellate lines proceeding from it, may be seen externally on the peritoneal covering.

In cases, however, where the ulcer has extended deeply, and where there has been great thickening of its base and margin, the contraction of the fibrous tissue, by which the loss of substance is replaced, may lead to alterations in the form and shape of the stomach. Amongst the most remarkable of these are cases when, from ulcers seated in the smaller curvature, the pyloric and cardiac orifices have been drawn into close proximity to one another;[2] or where the constriction extending around the centre of the stomach has given it an hour-glass shape, involving a special tendency to dilatation of the fundus;[3] or where the whole organ may have been found reduced to the size of the intestine.[4] Strictures also of the orifices are by no means uncommon, though much more frequent in the pyloric than in the cardiac extremity.[5] Extreme degrees of stenosis are rare (only about once in 200 cases—Brinton). When affecting the pyloric orifice, they are usually attended with extreme distension of the whole viscus, and are associated sometimes with thinning, and at others with thickening, of its muscular coats, while in a third class this dilatation is limited to peculiar pouch-like formations in the pyloric portion.[6]

Similar constrictions have also been observed in cases where cicatrization has ensued in ulcers situated in the duodenum, the effects of which on the stomach are very similar to those produced by narrowing of the pyloric orifice. Sometimes, however, dilatations, for which an explanation is not easily afforded, have been seen immediately beyond and below the constricted part (Krauss). The contractions may also lead to peculiar bending and twisting of the intestine, and in one case, recorded by Frerichs,[7] to further consequences due to the thickening of the tissues external to the bowel, resulting in complete obliteration of the vena portæ.

The contractions of the cicatricial tissue, and the change thence resulting, are, however, not always necessarily attended with closure of the ulcer, which in some of the cases just quoted has still been found open.[1]

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[1] Mr. Curling has given a case where cicatrization of an ulcer in the duodenum after a burn was found commencing on the tenth day; another when it was completed at the eighth week; and Mr. Holmes has recorded one where cicatrization in the duodenum was completed within twenty-eight days after the burn from which it had probably originated.

[2] As in a case by Bärnhoff, quoted by Henoch, loc. cit. 143, where the pyloric and cardiac orifices were only 1¼ inches distant.

[3] In cases by Cruveilhier, Budd, and Brinton.

[4] For a case of constriction of the cardiac orifice, with great consequent diminution of the size of the stomach, see Drasche, Wien. Med. Wochensch. 1854, No. 67 (Müller).

[5] Jaksch, loc. cit.

[6] See two cases by Cruveilhier, Anat. Path., Liv. liv. xx. Also a case quoted by Brinton, Dub. Med. Journ. ii. 494.

[7] Dis. of Liver, Syd. Soc. Trans. i. 272.

(*h*) *The extension* of the ulcer proceeds either by the destruction of the cicatricial tissue in its base and margins, or it may occur before this product has been formed. In the former case it is characterised by softening and liquefaction of the superficial layers of the base, while at the edges, where it is taking place, the thickened mass disappears, and the border presents the same sharply-cut limitation which characterised the first stage of the disease. In some instances this may be found affecting only part of the circumference, and thus affording a criterion of a return of the destructive process in an ulcer of old standing.

During its progress the larger vessels of the stomach are not unfrequently opened, and prove one of the sources of the hæmorrhage so frequently observed.

The rapid extension of an ulcer in the early stages of its formation is one of the most frequent causes of the accidents next to be considered.

(*i*) *Perforations* of all the coats of the stomach may be divided into two classes—namely, those in which an opening has ensued from its interior into the abdominal cavity, and those in which this event is prevented by adhesion to the surrounding viscera. The frequency with which the first-named of these events occurs has been calculated by Dr. Brinton, from 257 recorded cases of open ulcer, as being about equal to 13½ per cent. of all cases of ulceration; but, as this average is based upon an estimate of the comparative frequency of cicatrization, which there is reason to believe is considerably below that really found, so it is not improbable that the actual proportion of the cases in which this accident occurs is less than Dr. Brinton was disposed to believe;[2] though, as Cruveilhier has pointed out, it is much more frequent in cases of simple ulceration than in those of cancer of the stomach.

The influence exerted by age and sex on this event, having been already alluded to, does not appear to require further discussion. A very important feature in determining the character and nature of this result is to be found in the position of the ulcer itself in the wall of the stomach and duodenum. When this is situated in parts where the amount of movement and distension is the least, and where adhesions are most easily formed to surrounding parts, the probability of the accident is considerably less as compared with the cases where opposite conditions prevail;[3] and thus ulcers situated in the anterior wall of the stomach and on the lesser curvature are more liable to perforation than those in the posterior wall and the greater curvature; while, of the duodenal ulcers,[1] those situated in proximity to the head of the pancreas possess almost an immunity from this event.

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[1] See also a case of contraction of the duodenum by a recent ulcer, Duchek, Prag. Viertel-Jahresch. xxxvii. 51, 1853.

[2] Of Willigk's 231 cases of ulcers and cicatrices, as before quoted, only six had perforated; and Dittrich in 106 cases only met with ten cases of perforation. It is probable, also, as Miquel has observed, that in collections of isolated cases a greater number of instances of perforation will be found, owing to the remarkable character of the event conducing to its publication, while those of simple ulcer are not so commonly thought worthy of being recorded.

[3] Of 191 cases of perforation of the stomach by ulcers collected by Dr. Brinton, 69

Precise figures regarding the condition of the ulcer at the time of perforation are wanting; but, though the event is very frequent in those of old standing, there yet seems reason to believe that it is comparatively more so in the early stages of the disease, and before the protective thickening which subsequently ensues has had time to form. It seems not impossible that the depth to which the tissues are primarily affected by the process which gives rise to the slough, in which the ulcer often appears to originate, has no small influence in accelerating or retarding this event.

When it occurs in those of recent origin, or when there are no adhesions to neighbouring organs, the peritoneal covering gives way after the previous formation of an ashy-grey slough, the final yielding of which is generally determined by distension of the stomach, or by the effort of vomiting. The external opening is usually considerably smaller than the inner surface of the ulcer; its edges are generally sharp and well defined, like those of the rest of the ulcer, but sometimes they may be slightly ragged from the final rupture of the necrotized tissue. They are never, however, so ragged and thinned, nor do they present the same pulpy and transparent appearance as is seen in the case of perforation by post-mortem solution, from which the perforation is further distinguished by the characters of the alteration of the mucous membrane in the latter.[2]

When adhesions have formed to neighbouring organs, or when protective thickening has occurred in the sub-peritoneal tissues, the future progress of the ulcer is subject to many variations. Thus, it may deeply invade the tissue of the liver, spleen, or pancreas, giving rise to extensive hæmorrhage from their vessels;[3] or deep fistulous openings may form into the interior of these organs, in

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were on the lesser curvature, 55 on the anterior surface, 11 on the posterior surface, 9 at the pyloric extremity, 10 at the cardiac end, 4 in the middle of the organ, and "in no less than 24 there were two ulcers opposite one another on the anterior and posterior surfaces of the organ—the former being the site of the perforation, while the latter was in most instances firmly adherent to the pancreas." It is interesting to compare these returns with those of Jaksch relative to the comparative frequency of the sites of adhesion. These occurred in 22 out of 57 cases of ulcer, and in 15 they were between the posterior surface or lesser curvature of the stomach and the pancreas; in 5 between the pyloric portion or lesser curvature and the liver; while in one only there was adhesion to the mesentery, and, in one, to the spleen.

[1] Krauss states that, of 16 cases of perforation of the duodenum from this cause, in 12 the event had occurred in ulcers situated in the right wall of the upper horizontal portion.

[2] Cases are, however, recorded where the perforation took place, not at the seat of the ulcer, which had caused stricture of the pylorus, but from rupture of the fundus of the secondarily dilated stomach (Siebert, quoted by Müller, p. 110). A somewhat similar incident in a case of cancer of the stomach is recorded by Andral, Clin. Méd. ii. 75.

[3] Opening of the portal vein by a duodenal ulcer has been recorded by Rayer, Archives Gén. vii. 66; Andral, Prec. Path. Anat. ii. 177.

which also, in some cases, large abscesses may be found communicating with the ulcer; while, in the case of the liver, secondary abscesses are by no means uncommon.[1] In other, but rarer instances, perforations may occur in the diaphragm,[2] and the ulcer may give rise to pleurisy or to gangrenous inflammation of the lung, or may communicate with the bronchi; or perforations may ensue into the transverse colon,[3] or externally through the anterior wall of the abdomen.[4] Other events which have been recorded are erosions of the pancreatic[5] and biliary ducts, and sometimes, in the case of duodenal ulcers, the ducts have been found either in a state of suppuration,[6] or obstructed, thus giving rise to jaundice,[7] or even to bursting of the gall-bladder;[8] while, in other instances, perforation of this latter structure by the ulcer has been noticed;[9] in others the stomach has been so bent upon its axis, as to form a second direct communication with the duodenum through the adhesion and perforation of their walls thus brought into apposition, the pyloric ring remaining, in some cases, open and bridging the newly-formed aperture.[10]

(*k*) *Escape of the contents of the stomach into the cavity of the abdomen* is almost invariably followed by more or less general peritonitis,[11] associated with tympanitic distension, which sometimes proceeds to an extreme degree, owing to the escape of air through the opening in the stomach or duodenum (Abercrombie).

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[1] See some cases by Dr. Murchison, Path. Soc. Trans. xvii. 145.

[2] These, however, are generally caused by fistulous communications through adhesions with other organs, or with circumscribed abscesses. See cases by Dr. Habershon, Path. Soc. Trans. 1847-8, p. 252; Obs. Alimen. Canal, 1862, p. 82; Cruveilhier, Arch. Gén. de Méd. 1856, p. 155; also by Rokitanski and Jaksch.

[3] See a collection of the published cases of this nature in an able memoir by Dr. Murchison, on Gastro-Colic Fistula, Edin. Med. Surg. Journ. 1857-8. This perforation is much more frequent in cancer than in ulcer of the stomach; of 33 cases 9 or 10 only were from simple ulcer, 21 from cancer.

[4] Dr. Murchison has collected all the cases of Gastro-Cutaneous Fistula hitherto recorded in a memoir, Med.-Chir. Trans. xli. There is a striking contrast between these and Gastro-Colic Fistula in regard to the comparative frequency with which they are connected with simple and cancerous ulceration; for whereas the number of instances in which openings into the colon are associated with cancer greatly exceeds that in which they are caused by ulcers, Gastro-Cutaneous Fistula would appear to be nearly twice as frequently the result of simple ulcer as of cancer. This difference is probably due in part to the comparative rarity of simple ulcer "in that part of the stomach nearest the colon, whereas of simple ulcers ending in perforation a very large proportion is found in the anterior surface." Perforations externally caused by ulcer may be distinguished usually by the smoothness of the opening, while those from cancer are ragged and villous through the extension of the growth in the skin.

[5] Rokitanski; also Dittrich, Prager Viertel-Jahresch. xiii. 124.

[6] Förster, Würzb. Med. Zeitsch. 1861, ii. 158.

[7] Krauss, p. 21.

[8] Herzfelder, Wiener Zeitsch. 1846, p. 23 (Krauss).

[9] Dr. Barker, Lancet, June 1850, 776. Also Long, loc. cit. See also a case by Chomel (Andral, Prec. Path. Anat. ii. 116), where there was a fistulous communication between the duodenum, gall-bladder, and colon.

[10] Rokitanski, loc. cit. Cruveilhier, Rev. Méd. iii. 36. Dittrich, Prag. Viertel-Jahresch. xiii. In the latter case the communication took place through the pancreas.

[11] A remarkably exceptional case is recorded by Bardeleben, in which, though the patient lived twenty-four hours, and purgative medicines, together with other contents of the stomach, were found in the peritoneal cavity, there were no signs of general inflammation of its lining membrane. Virch. Archiv, v. 250.

In other cases however, where, as Dr. Brinton observes, a gradual filtering of the gastric contents has taken place through a small opening in its wall, or through incomplete adhesions, and particularly through those which are formed between the stomach and omentum, circumscribed abscesses, sometimes of considerable size, and communicating with the ulcer, may be formed. Their boundaries are then variously constituted by adhesions between the ulcer and the anterior wall of the abdomen, or with the diaphragm, liver, colon, pancreas, or spleen, which latter organs may be sometimes partially or completely destroyed; or the abscesses may be limited to the cavity of the small omentum, a variety of which Dr. Brinton has collected twenty instances. Such abscesses may be more or less permanent, but the adhesions may again give way; and this event is then followed by more general peritoneal inflammation.

In some cases where ulcers of the duodenum have opened into the loose cellular tissue bounding its posterior wall, the abscesses resulting have passed upwards through the mediastinum towards the neck,[1] or have opened outwards between the sixth and seventh rib on the right side, or, posteriorly, at the shoulder-blade.[2]

(*l*) *Hæmorrhages* from the ulcer require a short separate consideration.

Cruveilhier[3] divided them into slight, moderate, and excessive, and pointed out that those of the two first classes came from the smaller arteries and veins of the mucous and submucous tissue, which, when examined under water, can be seen either eroded and obstructed by recent and easily detached clots, or closed by more firmly attached coagula. Copious hæmorrhages may, however, sometimes be determined by venous congestion, since in some cases of this nature no other source has been found for the blood effused than varicose, dilated veins in the mucous membrane of the stomach.[4] The tendency is increased by hyperæmia, however originating, and especially by that occurring during the digestive act. It is probably also in this latter cause that an explanation must be sought for the cases where hæmorrhage has been increased or caused by moral emotions[5] or by the menstrual period.[6]

Larger hæmorrhages most commonly arise from the perforation of vessels of considerable size in the deeper coats. This event is indeed usually prevented by the coagulation of the blood in their interior, and by the resistant character of their walls; and it is by no means uncommon to find an obstructed vessel in the floor of an ulcer, in cases where no considerable hæmorrhage has taken place during life. The ulceration of the obstructed vessel, nevertheless, may proceed beyond the point to which the protective thrombosis has extended. In some cases extensive hæmorrhage has taken place in the early stages of ulceration, but in others the whole ulcer has been found cicatrized except at a point where an eroded artery, giving rise to fatal hæmorrhage, projected into its floor.[1]

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[1] Förster, Würzb. Med. Zeitsch. 1860, ii. 162 (Krauss).

[2] Gross. El. Path. Anat. 532. Also a case by Dr. Stretton, of Worcester, Lond. Med.-Phys. Journ. vol. lx. p. 43. In the latter instance there were openings in both the last-named situations, and food passed through the fistulous opening between the ribs five minutes after it had been taken.

[3] Archives Gén. 1856.

[4] Cruveilhier, Anat. Path., Liv. x. Also Frerichs.

[5] Ib., Liv. xx.

[6] Ib. Liv. xx.

Another source of both the larger and also of the smaller hæmorrhages is to be found in the penetration of the vessels of adjacent organs with which the ulcer has formed adhesions, and among these the splenic artery, which in its tortuous course often comes in contact with the stomach, affords a large proportion of instances of hæmorrhage. Perforations are, however, recorded of the portal veins and of the hepatic artery from duodenal ulcer.

When, on the other hand, hæmorrhage ensues from the main arteries of the stomach, it usually proceeds from those situated in the larger and smaller curvatures, and especially from the superior pyloric or coronary artery.[2] Moreover, as Miquel has also remarked, the presence of a large vessel at the base of an ulcer affords, by its resistance to the ulcerative process, a certain protection against the occurrence of perforation.

(*m*) *The condition of the mucous membrane* of the stomach offers also certain varieties. In many cases it is found perfectly healthy; in others, polypoid vegetations, or extravasations and hæmorrhagic erosions, may be found around the margins of the ulcer; signs also of chronic or recent catarrhal inflammatory action are not unfrequent. These latter are, however, most common when constrictions have occurred in the pyloric or duodenal portions of the canal. The appearances characteristic of these changes, and also the varicose dilatation of the vessels occasionally observed, do not, however, require further description in this place.

PATHOLOGY AND PATHENOGENESIS.—While some observers attributed these ulcers to inflammation, and others, as Cruveilhier, have expressed themselves unable to explain their origin, Rokitanski separated them from the class of ordinary inflammatory ulcerations, and declared that their point of departure was from a necrosis of the mucous membrane and subjacent tissues. Virchow[3] advanced

[1] Cruveilhier, Anat. Path., Liv. x. Also Boullay, Schmidt's Jahrbücher, vol. lxx. p. 185 (Miquel).

[2] Hence there is a certain contrast between the liability to perforation and hæmorrhage determined by the anatomical position of the disease in the walls of the stomach, since ulcers situated on the anterior surface, from which, as has been seen, perforation is most likely to ensue, rarely invade in their progress any of the larger vessels, whose branches are only sparingly distributed to this portion of the organ. Thus, of 52 cases of fatal hæmorrhage collected by Dr. Brinton, the ulcer giving rise to it was in 24 instances situated in the smaller curvature, in 17 on the posterior surface, in 6 on the pyloric extremity, and in 2 only on the anterior surface.

[3] See Handb. der Spec. Path. Therap. i. 256; Archiv, v. 362 et seq. Wien. Med. Woch. 1857, pp. 498, 499. Gesammelte Abhandlungen, p. 706.

the next step in this direction, by stating that the primary condition for their formation was an arrest of the circulation through a sufficient depth and area to permit the solvent action of the acid gastric juice to be exerted on the tissues deprived of the protective action of the alkaline blood. This view has received a direct confirmation from Dr. Pavy's[1] experiments on the effects of the arrest of the circulation in the stomach, which have shown that this event is invariably followed by the solution of the coats of the organ, proceeding in some instances to complete perforation; and it is still further corroborated by the case before quoted, where hydrochloric acid swallowed had given rise to an ulcer proving fatal by a perforation of the coronary artery.[2]

If this opinion be admitted as affording a probable explanation of many, if not most, of these cases, it follows that any causes capable of producing such an event may be competent to give rise to the ulcer in question; and among the most frequent of these appear to be extravasations of blood into the tissues of the stomach, to which a preponderant influence in this direction was first attributed by Rokitanski.

It appears, however, to be necessary that such extravasations should extend deeply, and occupy some considerable area, and that the minor petechial ecchymoses resulting from capillary hæmorrhage on the surface, and to which the name of hæmorrhagic erosions has been given, are, as a general rule, incapable of producing this effect, since the protective influence of the circulation is not, in these cases, sufficiently withdrawn, to permit of the action of the gastric juice extending deeply into the subjacent tissues.

Virchow states as his opinion, that the most frequent causes of this arrest of the circulation are obstructions of the arteries through embolism, extravasations through obstructions of the portal vein, fatty degeneration of the coats of the arteries, or even the diminished calibre of the vessels, sometimes met with in chlorosis, or extravasations caused by violent movements of the stomach in the act of vomiting.

The possibility of the direct production of these ulcers through embolism has been demonstrated by Panum's experiments. The immediate effect of the obstruction of the arteries thus produced was seen in extravasations into the submucous and mucous tissues, which were followed by ulcerations in these parts, having all the characters above described;[3] and Müller[4] has equally shown that very similar results may be produced by ligature of the vena portæ.[5] These observations would also explain some cases when acutely produced recent ulcers of the stomach and duodenum have been observed in pyæmic or metastatic affections, and possibly in the puerperal conditions to which allusion has been already made.[1]

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[1] Phil. Trans. 1868. A Treatise on the Function of Digestion, 1867.

[2] Note 7, page 915.

[3] Virchow's Archiv, xxv. p. 491 et seq.

[4] Loc. cit. p. 272 et seq.

[5] It is remarkable that cirrhosis of the liver does not more frequently give rise to ulcer of the stomach. Frerichs only gives three cases of the latter disease associated with disease of liver. Only one of these was associated with chronic atrophy. Förster gives a case of ulcer of the duodenum associated with cirrhosis, Würzburg Med. Zeitsch.

It has been remarked by Virchow that both the shape and also the most common positions of these ulcers point in many cases to their arterial origin, and that the conical form, with the base at the mucous surface, corresponds to the ramifications of the capillary branches spreading outwards from a main trunk, which has been obstructed in the deeper tissues, and thus resembling precisely the results of arterial embolism in other parts where similar appearances are seen.

The appearances of extravasations found in connexion with some recent ulcers, which have been before alluded to, seem also in a great degree to confirm this theory of their hæmorrhagic origin. It may further be noted that many of the causes to which their production has been attributed are such as would coincide with this opinion, though their inconstancy in this respect is easily explicable, when we consider that though the tendency of the majority is to produce congestion of the stomach and intestines, yet that this may proceed to a marked degree without necessarily involving the occurrence of such extravasations as are capable of giving rise to extensive or deeply-spreading necrosis of the coats of these organs. Some clue may thus be gained to their frequent appearance in connexion with disturbances of the menstruation, which are known to give rise to great disorder in the functions of the stomach. This is further evidenced by the hæmorrhage from this organ, which occasionally replaces that from the uterus, and which must necessarily have been preceded by an extreme hyperæmia, not only of its lining membrane, but also of all the vessels supplying it; while the not unfrequent coincidence of the disease with the puerperal state may be attributed either to conditions of congestion, or, probably in many, to embolism associated with pyæmia.

A similar origin may also with great probability be ascribed to duodenal ulcers commencing after burns of the skin, though it is possible that the direct mechanism of their production may be by means of embolic processes rather than through simple congestion. Their site in these cases however appears to require further explanation, since the possible implication of Brunner's glands, suggested by Messrs. Bowman and Curling, does not appear completely to elucidate their peculiar frequency in this position under these

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1861, ii. p. 165 (Krauss). A case is recorded by Gunsburg, Arch. Phys. Heilk., where the determining cause of the ulcer appeared to be the obstruction of a vein in the stomach itself.

[1] See a case by Buhl, Bericht über 280 leichen Oeffnungen, Henle and Pfeuffer's Zeitsch. Rat. Med. 3d. ser. viii. 1857, p. 34, where there were recent perforating ulcers of the duodenum and intestine in a case of pyæmia associated with parotid abscess. See also a case of gangrene of the intestine from embolism. Gaz. Hebdom. 1862, Oct. 3.

circumstances as compared with their relative rarity under other conditions. The appearances of recent extravasations observed in several recorded cases of this nature may very properly be placed in juxtaposition with Rokitanski's observations on the frequency of hæmorrhage from the lower portions of the intestinal canal in connexion with similar causes, as pointing to their origin in intense congestion leading to rupture of the vessels.[1] A similar explanation may thus be afforded of an instance recorded by Cruveilhier,[2] when the symptoms of ulceration followed an attack of cholera; for although in this disease hæmorrhage is more common in the intestines than in the stomach, yet extravasations in the latter are also occasionally observed.[3]

Dr. Copland's independent testimony[4] confirms Virchow's opinion that extravasations having a similar result may arise through atheromatous degeneration of the coats of the vessels of the stomach. Frerichs, also, in a case where an ulcer of the stomach followed the obstruction of the gall ducts,[5] has pointed out that Kölliker and Müller[6] observed the disease in the duodenum in cases where the common bile duct had been ligatured, and where also calcareous matter was deposited in the branches of the cœliac axis and in those of the mesenteric arteries.

Our knowledge of other possible causes of the disease must be considered as yet imperfect.[7] It may be questioned whether inflammatory states often, if ever, exist in the stomach to a degree sufficient to act in a manner similar to the conditions which have now been described. It appears improbable that the follicular ulcerations proceeding from the solitary glands of the mucous membrane often extend beyond the area occupied by these structures, though the distribution of anatomical elements of a lymphatic character has been shown, by Dr. H. Jones's and the author's observations, to extend more widely in the mucous and submucous tissue, than has been sometimes supposed.

Another more likely cause, though of rarer occurrence, is found in those cases where profuse suppuration takes place in the submucous tissue, the effects of which are extremely likely to arrest the vascular supply of the mucous membrane.[8]

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[1] Mr. Erichsen had previously pointed out only the probability of the extreme congestion of the gastro-intestinal canal acting as the cause of these lesions.

[2] Rev. Méd. 1838, iii. 32. Path. Anat., Liv. xx.

[3] See author's report on Stomach and Intestines in Cholera. Path. Soc. Trans. 1867. Also Cruveilhier, Path. Anat., Liv. xiv. Also Buhl, Report of Munich Commission on Epid. of Cholera, 1854, p. 500.

[4] Med. Dict. vol. iii. pt. 2, p. 919.

[5] Dis. Liver, Syd. Soc. Trans., by Murchison, i. 137.

[6] Würzburg Verhand. vi. 474.

[7] An experiment of Frerichs, Dis. Liver, i. 187, would lead to the question whether disturbances of the circulation or of the nutrition of the stomach through derangement of the nervous supply may not occasionally produce this effect. He found an ulcer of the stomach in a cat after division of the splanchnic nerves and of the cœliac axis.

[8] See Dittrich, quoted by Brand, Ueber Stenosen des Pylorus, Diss. Inaug. Erlangen, 1851; also Reynaud on Gastritis Submucosa, Bull. Soc. Anat. 1861, 2d ser. vi. 89, cases quoted of this possible origin of ulcers.

It is also not impossible that excessive secretion, or excessive acidity of the gastric juice, particularly in the absence of food in the stomach, as when the secretion is excited by alcoholic drinks, may also give rise to ulceration, since it has been shown that an excess of acid introduced into the stomach may produce similar effects.[1]

However originating, the peculiar tendencies to extension and perforation seem to be especially communicated to these ulcers by the action of the gastric juice; for though ulcerations of the lower portions of the intestines are very common in other diseases, perforation is a very rare event, unless caused by the direct necrosis of the new formations of tubercle or typhoid. It is also worthy of remark that ulcers of the kind now described are, with rare exceptions, limited in their appearance to the upper part of the canal, where the solvent power of the gastric juice is still active, that of the intestines on protein substances being very inferior in this respect; and though the causes of ulceration may affect equally the whole canal, these special effects are only produced by the additional influence thus exerted.

Symptoms.—The symptoms which are characteristic of this disease have been enumerated as pain, vomiting, hæmorrhage, disturbances of the digestion, and alterations of the secretions of the stomach. The whole of these are not, however, constantly present in any single case, and there is a considerable diversity observed in their relative predominance, and in the order in which they may appear; while in a certain number, and especially in those which run a rapid course, leading to perforation, the disease may have been entirely latent until within a few hours of a fatal issue.

Pain may be regarded as the most constant of these symptoms, so much so that in its absence the diagnosis of gastric ulcer would scarcely be justified during life. It is often the earliest in its appearance, though in some recorded cases it has been preceded, for a longer or shorter period, by disturbances of the digestion, and also by pyrosis, or by excessive secretion of acid supervening immediately after meals.

There are considerable differences, however, observed in its character, intensity, duration, and mode of accession, which appear to be in some measure explicable by the extent of the ulcer, by the implication of large nerve trunks, and by the manner in which, from its position, it is affected by the movements of the stomach.

It seems, however, not unimportant to observe that the varieties of pain described, though not in all cases absolutely distinguishable, may be divided into two classes. The first of these is more or less constant, and its characters are described as those of wearing, burning, boring, but rarely (Brinton) sharp and lancinating; often confined to a very limited space at the epigastrium, but felt also in the back behind the lower dorsal and first lumbar vertebræ, and between the scapulæ, in both of which latter situations, as remarked by Cruveilhier, the pain may sometimes exhibit a greater intensity than at the epigastrium. It is sometimes felt also towards the hypochondria or umbilicus, and deviations in these directions have been found by Dr. Brinton to accord with corresponding variations in the site of the ulcer. It is generally increased, and only in rarer cases relieved, by taking food. The second variety tends to occur in paroxysms, and is felt over a more extended area. It is of intense severity, so as sometimes to cause fainting, or even convulsions (Bamberger). The attacks, which are usually described as cardialgia, are often attended with strong abdominal pulsation, probably due to a reflex paralysis of the muscular coats of the abdominal aorta.

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[1] See notes 6 and 7, p. 915.

The more constant pain, which in many cases, when exacerbations are absent, consists rather in a sense of uneasiness than of more acute suffering, seems to be directly referable to the existence of the ulcer, and is felt with a severity proportioned to the depth to which it has extended, and especially to the implication of the peritoneal surface of the organ. The paroxysmal form is generally relieved by emptying the stomach of its contents by vomiting: hence in many cases it is probable that it is due directly to the irritating effects of food, and to the movements of the stomach caused by its presence, and also to distension by flatus, or to the generation of abnormal acids by fermentation, most of which causes act with greater intensity when adhesions have been contracted to surrounding parts.

These paroxysms have also been met with when large branches of nerves[1] have been invaded by the ulcer; and as their frequency and intensity have in some cases been found to correspond with the occurrence of hæmorrhage, and also with the size of the ulcer, it is not improbable that they are in some measure connected with its extension. They are also not unfrequently caused by moral emotions, and by exposure to cold and wet, or by bodily exertion; so that, although in the majority of cases they are explicable by the physical condition of the stomach, a certain class remains in which the paroxysmal character must be referred to special conditions affecting the nervous system; and some instances are recorded by Traube[2] when during these attacks there was hyperæsthesia or anæsthesia of the cutaneous surface of the thorax, and even pain extending down the arm.

Complete intermissions of pain, sometimes extending over a considerable period, are occasionally observed; or there may only remain in these intervals a slight degree of epigastric uneasiness, somewhat increased by taking food. It is a not improbable inference that the relief thus experienced may be due to periods when a cicatrization of the ulcer is taking place, since the converse condition, viz. its extension, appears to be associated with periods of aggravation.

Aggravations of the pain have been observed to precede the menstrual period, and to diminish in intensity on the establishment of the discharge.

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[1] Habershon, loc. cit. 129, 131.

[2] Deutsche Klinik, 1861, p. 63.

The pain is almost invariably[1] aggravated by pressure, and there is frequently such a degree of epigastric tenderness that the slightest pressure from the clothes, or the gentlest touch by the hand,[2] becomes unendurable. The amount of tenderness depends in some degree on the position of the ulcer, those situated on the posterior surface being less affected by manipulation. In some cases an increase of the dorsal pain has been observed to follow pressure on the epigastrium.

The effect of food is also most invariably to bring on or to aggravate pain already existing. The period at which the pain follows its ingestion varies in different cases, and in some it has been observed to depend on the site of the ulcer, those in the neighbourhood of the cardia or fundus being often more speedily followed by this symptom than when the disease is situated in more distant parts of the organ. It is seldom, however, that its appearance is long delayed; and in fact the early supervention of this symptom under these circumstances is one of the most valuable diagnostic criteria of the nature of the complaint.

When pain occurs at longer intervals after food, as after a lapse of one or two hours, it is in all probability attributable either to flatulent distension of the stomach, or to excessive acidity, or in some cases to the position of the ulcer at the pyloric orifice, or in the duodenum, when the passage of the food from the stomach brings on an aggravation of suffering. It does not appear, however, that this difference can be depended upon with any certainty as a test of the position of the ulcer, for the symptom may be early excited, whatever the site of the disease; and, secondly, food passes out of the stomach, though in comparatively small quantities, at a very early period after its introduction.[3]

Variations in the intensity of the pain have been observed to depend on the position assumed by the patient being such as to free the ulcer from contact with the contents of the stomach.[4] This test also is not, however, infallible, as in some cases the severity of the pain, whatever the side of the ulcer, is unaffected by posture, and in some it has even been relieved by the patient lying or pressing towards the seat of the disease.[5]

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[1] Exceptional cases are recorded by Abercrombie, Henoch, Cruveilhier (Path. Anat., Livr. x., xx.), and by Miquel (Van Deen, Schmidt's Jahrbücher, li. i. 39), where strong pressure on the front of the abdomen gave relief. In one of Cruveilhier's cases the ulcer was situated on the posterior wall, in another on the anterior surface. Miquel advances in explanation of these cases the probable hypothesis, that the mitigation thus experienced, which was particularly observed during paroxysmal attacks, may be due to the restraint thus exercised on the movements of the stomach.

[2] The necessity of caution in this proceeding, to avoid rupture of the stomach, requires to be insisted upon.

[3] See Dr. Stretton's case, before quoted; also Busch's observations on a case of duodenal fistula. Virchow's Archiv, xiv.

[4] First observed by Dr. Osborne, Dub. Med. Journ. xxvii. 361. Dr. Brinton says that two-thirds of the cases which he personally observed exhibited a marked influence of posture on the pain.

[5] See Brinton, Ulcer of Stomach, p. 71. Also Chambers, Lond. Journ. Med. 1852, and Nasse, Schmidt's Jahrbücher, 72, who found an ulcer of the anterior surface relieved by a prone decubitus.

Though no small share in the production of the pain by food is probably due to the movements of the stomach and to the acid secretion excited by the food, yet it is found that in the majority of instances its severity is increased by indigestible and stimulating substances, and by hot liquids. Some very exceptional cases are, however, recorded, not only in which bland articles of diet have given relief—a more common occurrence—but also where stimulants, and even brandy, have sometimes mitigated severe pain.[1]

Vomiting stands next to pain in order of its frequency and importance. It is, however, sometimes absent throughout the whole course of the disease, while in other cases even the blandest articles of food are immediately rejected. Ulcers situated in the neighbourhood of the pylorus seem to be more constantly associated with this symptom than those found in other parts. In some cases it appears as a mere regurgitation of the food, without much nausea or straining; and it usually attends and sometimes terminates the paroxysmal attacks of pain.

The matters vomited vary; food is returned altered in proportion to the time during which it has remained in the stomach. Acid or neutral fluids are also brought up, sometimes in considerable quantities, either independently of food, or, when accompanying it, far exceeding the amount of fluid which has been swallowed. In other cases evidence of the destruction of adjacent organs has been found in the vomited matters, as liver structures[2] and the elastic fibres of the spleen.[3]

The sarcina ventriculi is occasionally, but not frequently, seen in the vomited matters.

Hæmorrhage is generally revealed by vomiting; when however small quantities of blood are effused, it is possible that they may often escape notice, and therefore the frequency of this symptom cannot be accurately estimated. Of larger hæmorrhages, Miquel has found fifteen instances in ninety-one cases, while Brinton thinks that they occur in about one-third of all the cases of ulcer. In a case recorded by Cruveilhier[4] the hæmorrhage from the stomach frequently recurred at the menstrual periods, and sometimes replaced this discharge.

The characters of the blood brought up differ with the amount effused, and also with the rapidity with which it has escaped. Smaller quantities have usually a black or coffee-ground appearance, which may only appear in striæ, or patches on the mucus or food by which they are accompanied. Larger hæmorrhages poured out quickly from arteries of considerable size may, when vomited, still retain an alkaline reaction and arterial colour; while in other cases the blood is coagulated, and more or less blackened by the action of the gastric juice.

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[1] See a case by Mr. Travers and Dr. Farre, Med.-Chir. Trans. vol. viii. Several cases of this kind, chiefly from foreign authors, are also quoted by Miquel, loc. cit. p. 16; in some of them the diagnosis of ulceration was verified by post-mortem investigation. See also Duchek, loc. cit.

[2] Miquel, loc. cit. p. 25.

[3] Sangalli under Virchow, Schmidt's Jahrbücher, 1854, iv. 45.

[4] Path. Anat., Liv. xx.

In some instances, however, none of the blood effused is vomited, but the whole escapes by the bowel. The blackened and tarry appearance which the evacuations present under these circumstances, and the distinguishing features of its origin, will be hereafter described (see Hæmorrhage). In a third class, the hæmorrhage may at once prove fatal by causing syncope, without previous vomiting, and the cause of death may only be revealed *post mortem* by the distension of the stomach and upper part of the small intestines by large quantities of blood.

Certain prodromata may precede or accompany the hæmorrhage. The chief of these are a sense of heat, pulsation, fulness, weight or load at the epigastrium. They are not, however, commonly prominent, and are frequently unmarked, and the expulsive act is rarely preceded by any long-continued nausea. The prostration which follows excessive bleeding is sometimes very serious and alarming. The patient may be completely blanched, and the slightest movement may threaten syncope. When this condition persists without vomiting, a sedulous examination of the stools is necessary, to avoid the possibility of overlooking a loss of blood which is still proceeding, but which is only revealed by such an investigation. Blood thus escaping by stool sometimes causes diarrhœa, at others, griping and colicky pains.

The digestion is liable to be influenced by various circumstances, which form very complex features in estimating the influence of this particular disease upon the functions of the stomach, and it is difficult to separate its derangements from the pain to which the mere contact of food with the surface of the ulcer gives rise. When the mucous membrane is but little affected, it may proceed almost unimpaired. The mere presence, however, of the ulcer generally brings with it conditions of derangement which are seldom without an unfavourable influence, through the effects of adhesions impairing movement, or through the direct influence of pain, or through the invasion of the nerves by the ulcer either directly impeding the secretion of the gastric juice, or giving rise to various abnormal alterations of its quality. The evidence of this latter state is often the most prominent, and pyrosis of an acid or alkaline character may constitute for years the chief symptom present; but few cases escape without other dyspeptic derangements, and flatulence is a very common symptom. In other instances the digestion appears to be simply delayed, and to be attended throughout by a sense of epigastric load and uneasiness, and with eructations which are very liable to pass into vomiting. These symptoms are also largely influenced by the nature of the food taken; and though idiosyncrasies[1] exist, in this respect forming exceptions to the rule, they are usually aggravated by indigestible substances of all kinds.

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[1] See some cases by Oppolzer, Wien. Med. Woch. 1851, where raw ham agreed well.

When pyloric obstruction has been caused by an open or cicatrized ulcer, these symptoms are greatly aggravated. Nor does the healing of ulcers situated in other parts bring complete alleviation, as the contractions thence resulting may greatly interfere with the movements of the organ; and long persistent dyspepsia has been traced to this cause.

The appetite often suffers but little; frequently it is excessive and ravenous—a condition explicable in some instances by the loss sustained through vomiting, but in others probably to be referred to perverted innervation. When severe pain is present, it is often diminished. In some of these cases patients desire to eat, but fear to do so on account of the pain excited by food; in others a true anorexia is present.

The appearance of the tongue is probably influenced in no small measure by the condition of the mucous membrane of the stomach. In many cases it presents no deviations from that of health; in others it is more or less furred, or red and fissured. Sometimes during exacerbations of the disease it becomes aphthous (Abercrombie and Henoch). It may, however, be stated, that as a general rule it offers no distinct indications either of the presence or nature of the disease.

An excessive secretion of saliva,[1] in which the sulphocyanide of potassium is remarkably deficient (Bamberger), has been not unfrequently observed.

Constipation is a very constant symptom. It is probably in some cases due in part to the vomiting; but some influence may also be ascribed to reflex impairment of the intestinal action from the presence of the disease: in other instances, as in one recorded by Dr. Budd,[2] it is directly due to adhesive peritonitis glueing the coats of the intestines together, a condition which can scarcely be supposed to exist without some degree of simultaneous muscular paralysis. In some cases the constipation has a tendency to increase the vomiting, which then may be relieved by acting on the bowels by means of enemata.

The general strength of the patient often suffers in an extreme degree in the later stages of the disease. When vomiting is a prominent symptom, and when hæmorrhage and severe pain are superadded, a cachectic condition is induced, which it is exceedingly difficult, if not impossible, to distinguish, *per se*, from that of cancer. There is often the same earthy tint of skin: and in females, in whom the menstruation is disturbed, this may exist to a very marked degree. Others have after hæmorrhage the waxy look of extreme anæmia; and the latter appearance is extremely common in young girls who are the subjects of the disease.

Cachexia, however, except when hæmorrhage is present, does not usually occur early in the disorder; and when vomiting and hæmorrhage only take place at long intervals, both it and emaciation may be absent during many years in which the disease has, in all probability, existed.[1] Cases also are met with in which, though presenting very characteristic signs of the disease, no *external* appearance of departure from sound health is observable, while in others, which have run an almost latent course, the only symptom observed has been a gradually progressing emaciation.[2]

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[1] Osborne, Dubl. Med. Journ. xxvii. 365.
[2] Loc. cit. 128.

In other instances, as was observed by Jaksch, the disease appears to run an acute febrile course, with dull pain at the epigastrium, nausea, and vomiting, a loaded tongue, on which aphthæ are observed, with complete anorexia. The fever is more or less continuous; the skin is hotter than natural, and the cheeks are flushed. This condition, of which I have observed some instances, tends especially to occur during periods when pain has been more than usually severe. Its appearance may probably be accounted for either by the supposition of an inflammatory condition of the rest of the mucous membrane of the stomach, or, in the cases in which this symptom has appeared towards the close of life, when the patients are exhausted by the duration of the complaint, it may probably often arise from some intercurrent inflammatory action, and especially from pneumonia, which is found to be a common complication.[3] The frequency with which tuberculosis is met with in cases of the disease would also probably serve to explain the febrile reaction in such instances.

The frequency of the connexion of amenorrhœa with the gastric ulcer has been treated of in the etiology of the complaint.

The symptoms of duodenal ulcer differ but little from those which are met with when the disease occurs in the stomach.

Pain is often a less prominent feature, its absence being explained by Dr. Budd to depend on the fact that this part of the canal is subjected to less movement than the stomach. It may, however, exist with the same severity, and with all the features which have been before described; and, as in the case before quoted, it has been known to occur when the stomach is empty.[4] It has been observed in some cases to be limited to the right hypochondrium, and to be associated with marked tenderness on pressure in the same region. In a case recorded by Mayer an excessive feeling of hunger was noticed.[5]

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[1] A case by Henoch, loc. cit. ii. 122.

[2] Lees, Diseases of the Stomach, pp. 73, 74.

[3] In the case before alluded to, where the ulcer was caused by the swallowing of hydrochloric acid, there was a second extension of the ulcer accompanied by a marked secondary series of pyrexial phenomena. A boy æt. 11 swallowed ℥j. of "spirits of salt" on the 30th May. Admission to hospital on the 1st June, with pain in stomach and vomiting of blood-stained mucus. Pain and pyrexia, with a temperature not exceeding 100·4 Fahr., continued until June 5th. From this date the symptoms ceased, and the temperature remained normal until June 10th, when copious hæmatemesis occurred, followed by increased pain and tenderness. June 11th, temp. 99·2; June 12th, temp. 104·2; June 13th, temp. 101·2; June 14th, copious hæmatemesis, proving fatal within a few hours. There were adhesions of the stomach to the transverse colon and liver, but no other signs of peritonitis. This ulcer was situated about 1½ inches from the pyloric ring, and had perforated a large artery. No other condition could be found to account for the pyrexia.

[4] See also a case by Mayer, loc. cit. 106.

[5] The same fact was observed in Busch's case of duodenal fistula.

Vomiting is also said by Krauss to be less frequent, and when present it is generally associated with stricture of the intestine. It sometimes occurs in connexion with attacks of cardialgia, when its explanation is probably to be found in a reflected irritation communicated to the stomach.

Hæmorrhages, revealed by vomiting or by the state of the fæces, occur in about one-third of the recorded cases; and fatal cases, when death has taken place immediately by syncope, have also been recorded.[1]

The disease in this region also frequently appears to run a latent course, unrevealed by any symptoms beyond those of comparatively slight dyspeptic derangements, until fatal perforation suddenly occurs.[2]

Dr. Brinton has stated that diarrhœa is common in ulceration of the duodenum, and that it thus forms a contrast with the constipation observed when the stomach is the seat of the disease. Krauss, however, who appears to have collected the largest number of cases of this disorder, regards diarrhœa as being only an exceptional symptom, and says that constipation is the rule in the duodenal, as in the gastric, ulcer.

The symptoms of perforation, both of gastric and duodenal ulcers, are almost invariably sudden in their invasion; but in a few cases, probably where perforation has been gradual, they have been observed slowly to increase in intensity; and a similar course is witnessed in instances accompanied by the formation of local abscess in the cavity of the abdomen.

In the first class, however, which forms much the largest number, they usually supervene after a full meal, or after some violent exertion, or after vomiting, or the effort at defæcation.[3] They are ushered in with an intense pain in the abdomen; sometimes a sensation of tearing has been described (Dahlerup); rigors have also been noticed at the outset. Severe general abdominal pain follows, which is greatly aggravated by vomiting or by severe retching. An intense degree of collapse is frequently associated with these symptoms; the face is pale and sunken, anxious, and hippocratic; the pulse small, rapid, and almost imperceptible; the limbs cold and tremulous; suppression of urine has been also observed,[4] and death may take place suddenly at this stage from shock.

If life is prolonged, the symptoms which follow are those of general peritonitis. The abdominal muscles are at first spasmodically contracted and drawn into knots (Crisp)[5]; subsequently the abdomen becomes greatly distended from gas escaping into its cavity, and the percussion note is uniformly tympanitic. The liver is also pushed backwards, so that its dull resonance in front is replaced by a tympanitic percussion note under the ribs (Oppolzer). Respiration is entirely thoracic and sighing, the knees are drawn up on the abdomen, and the patient is afraid to speak or move through fear of increasing his sufferings: the abdomen is also intensely tender on pressure. If life is sufficiently prolonged, signs of an accumulation of fluid may be detected in the lower part of the cavity; but death usually takes place in a few hours, or at most after two or three days.

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[1] Cas. Broussais, Duodénite Chronique, 1825, p. 65.

[2] See some cases of this nature by Dr. Budd. Also one by Dr. Murchison, Path. Soc. Trans. ix. 198.

[3] Bouillaud, Arch. de Méd. i. 534, cited by Andral. Pressure on the epigastrium, through leaning out of a window, is mentioned as a cause by Henoch.

[4] Dr. Sedgwick, Lancet, June 15, 1867.

[5] Also noted by Cruveilhier.

In other cases, where circumscribed abscess has formed, the signs of general peritonitis may be wanting, and those of the localised inflammation may be more or less distinct, in the form of limited pain and tenderness, associated sometimes with circumscribed enlargement, and even with a distinct fluctuating tumour. In these cases symptoms of general peritonitis may follow at later periods, owing to the escape of the contents of the abscess into the abdominal cavity. Perforation of other structures may be shown by a fistulous communication with the external surface, or by signs of empyema or pneumothorax, or by a large expectoration of puriform fluid, associated with more or less hæmoptysis, or by the physical signs of a cavity, or of circumscribed gangrenous pneumonia at the base of the lung, or, when the colon had been invaded, by the passage of undigested matter by stool soon after food has been taken, or by fæcal vomiting,[1] and a fæcal odour of the breath.

COURSE AND DURATION.—Both the course and duration of these ulcers are, as will be seen from the foregoing description, very variable. Two main classes may, however, be appropriately established; one, when the disorder is of short duration, tending either to an early cicatrization, or to a rapidly fatal termination by perforation or hæmorrhage, and another, when the disease is of almost indefinite duration, lasting with intermissions during many years,[2] and occasionally interrupted by severe attacks of pain, vomiting, or hæmorrhage—variations which may probably be explained by the occurrence of imperfect cicatrization alternating with renewed extension of the ulcerative process.

PROGNOSIS.—Cruveilhier's statement, that "the simple ulcer of the stomach tends essentially to a cure," is fully confirmed by the number of cicatrices found *post mortem* as compared with that of open ulcers. Additional support is afforded to a hopeful prognosis by recorded cases,[3] where, after long persistence of the symptoms of ulcer, these had subsided, and the presence of cicatrices has been revealed by autopsies made after death from other causes, and sometimes after long intervals of time. It cannot, however, be denied that the dangerous nature of the events which may occur during its course contribute to impart to the disease a character of extreme gravity.

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[1] This has been recorded in only one case by Abercrombie.

[2] Dr. Brinton quotes cases where evidence of continuous disease had lasted in one 35 years; in two 30; three or four 20; in four or five 15; and in several 10, 7, 5, and 4 years. Dr. King Chambers has also recorded a case (Indigestions, p. 185), where the patient died of ulcer of the stomach thirty years after the first hæmatemesis. Other instances are given by Cruveilhier and Dr. H. Jones.

[3] Cruveilhier, Archiv. Gén. 1856, p. 160. The case of the celebrated anatomist Beclard, given by Billard, De la Membrane Muqueuse Gastro-Intestinale, 1825, p. 558, is an interesting example of this fact. After severe intellectual labour he suffered from pain at the stomach and vomiting; but by a careful diet, local bleeding, and counter-irritation these ameliorated, though only gradually. After his death, many years later, the cicatrix of an ulcer was found in the small curvature of his stomach.

Even the periods of comparative immunity from the more urgent symptoms by no means, in all cases, justify the conclusion that the disease is at an end, for intermissions of these followed by renewed exacerbations are not uncommon. The hope of a permanent cure diminishes also in proportion to the length of time which the disease has lasted; the cicatrization in these cases being impeded by thickening of the margins of the ulcer, by the implication of other organs, and by the inversion of the mucous membrane into the funnel-shaped excavation.

When a cure is not effected, the progress to a fatal termination is generally slow, sometimes extending through periods of many years; the patient dying finally of exhaustion and marasmus induced by the pain, vomiting, hæmorrhage, and disturbances of digestion.

In other cases, as has been before described, life may be immediately cut short by hæmorrhage or perforation. The risk of the former of these accidents is not very considerable, amounting, according to the estimates of Dr. Brinton (with which those of Müller and Miquel pretty closely correspond), only to about 3¼ to 5 per cent. of all cases.

The frequency of perforation has been stated to be open to discussion; but, under the most favourable estimates, the danger of death from this cause is considerably greater than that from hæmorrhage. The prognosis in this respect is liable to be influenced by the age of the patient, for it has been seen that with advancing years the liability to this event is considerably diminished. The risk in the early periods of life appears also to be greater in the female than in the male sex.

Even, however, in the commonly fatal event of direct rupture of the stomach and escape of its contents into the cavity of the abdomen, life need not be absolutely despaired of, since cases have been brought forward which prove that recovery may take place even after this has ensued.[1]

After the formation of an external fistula through the abdominal parietes, life in some cases seems to be prolonged without much suffering, and cases of cure by closure of the external opening have been recorded.[1]

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[1] See a most interesting case of this nature by Dr. Hughes and Messrs. Hilton and Ray, Guy's Hosp. Rep. 2d Series, vol. iv., of a girl in whom all the symptoms of perforation occurred, but ended in recovery. At a later period, after a meal which had greatly distended the stomach, the same patient was again attacked in a similar manner, and death ensued. At the autopsy two open ulcers were found in the stomach, one of which had perforated. There was a cicatrix of a former ulcer, and old adhesions existed between the stomach and adjacent viscera, and also between coils of the intestine, giving evidence of a previous attack of peritonitis. Miquel relates a similar case of recovery, but not verified by post-mortem examination. Another of the same kind is given by Dr. Hughes Bennett, Clin. Med. 487.

Even with the completion of the process of cicatrization the cure of the patient can hardly be said to be perfect. The cicatrix by involving branches of nerves may be the source of long-continued pain, and probably also of derangements of the gastric secretion, while alterations in the shape of the organ, arising from the healing of large ulcers seated in its central portions, may often, by interfering with its movements, prove the source of permanent disturbance of the digestion. The evils resulting from contractions, from the same cause, of the pylorus and duodenum will be again alluded to. Nor can the risk of a renewal of the ulcerative process in the tissue of the cicatrix be lightly passed over, since many cases are recorded in which the disease has returned in its original seat, and ended in perforation or hæmorrhage after the ulcer had been apparently closed, or, at least, after all the more urgent symptoms had ceased for years; a liability which induced Cruveilhier to express the opinion that both these accidents are more liable to occur "consecutively,"—*i.e.* by the erosion of the cicatrix,—than "primitively" during the period of the formation of the ulcer.[2]

Nor can another liability be forgotten, of which proof is afforded both by the multiplicity in some cases of open ulcers, and by the frequent co-existence of these with cicatrices,—viz. that the causes inducing the disease tend to remain in operation in the same individual, and, that though one ulcer may have healed, another may be formed and may prove fatal at a subsequent period.

DIAGNOSIS.—The symptoms of ulcer of the stomach require to be distinguished from the severer forms of neuralgic affection, from some cases of chronic inflammatory action, from cancer of the stomach, and in some cases from colic. The distinguishing features of the affection are severe localised *persistent* pain, intensified in paroxysms, aggravated by food, and associated with tenderness on pressure, with vomiting, and with hæmorrhage, disclosed either by vomiting or by stool.

Without the simultaneous occurrence of the greater number of these symptoms, the diagnosis of ulcer must often remain somewhat uncertain, and it is the combination now enumerated which must mainly serve as the basis of diagnosis.

Some of them, however, are more frequent than others, and localised pain aggravated by food and associated with localised tenderness is sufficient to excite the gravest suspicions of the nature of the disease.

In another class, however, persistent dyspeptic symptoms associated with extreme degrees of acidity, unaffected by food or by treatment, have proved the sole symptoms of the disorder, which has ended fatally by perforation.[1]

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[1] Dublin Journal, vi. 148, from an American source; Western Journal Med. Phys. Science, 1834. Middeldorp has also almost completely succeeded in closing a gastric fistula by a plastic operation, Canstatt's Jahresb. 1859, iii. 187, Brit. For. Rev. Oct. 1860.

[2] Arch. Gén. p. 160.

The diagnosis from *chronic catarrhal inflammation* is as a rule only difficult in those cases of the latter disorder which are attended with hæmatemesis from congestion. The distinctive features of this form of disorder have been already described (see Chronic Catarrh, pp. 900, 906). The other forms of chronic catarrhal inflammation are rarely associated with distinct or severe gastric pain. Vomiting also is a much rarer symptom. They are, further, usually associated with more marked symptoms of dyspeptic derangement, with a more loaded tongue, and with more thirst, malaise, and pyrexia than are commonly observed in cases of ulcer.

The chief features which distinguish *ulcer* from *neuralgic affections* of the stomach have been already passed in review; it may be stated, in addition, that the aggravation of the pain by pressure, when tenderness of the muscles can be excluded, is another most important means of diagnosis. It is rarely absent, unless in cases when the ulcer is situated in the posterior wall of the stomach, and even then it can usually be elicited on deep pressure; and the exceptional cases in which the pain of ulcer has been noticed to be relieved by pressure are scarcely sufficiently numerous to invalidate a diagnosis founded on these data. The tenderness in ulcer also exists in the intervals of the paroxysmal pain. Aggravation of the pain at the menstrual period in females is of less value, as it has been observed both in cases of neuralgic origin and also in ulcer. Exceptional cases may, however, occur in the vomiting from cerebral disease, when epigastric pain, tenderness, and hæmatemesis have all been present, but when no lesion has been found in the stomach after death.[2] They are fortunately however, of extreme rarity, since the criteria for an accurate diagnosis are in such instances almost entirely absent. The danger, however, consists rather in overlooking the cerebral condition, than of mistaking the nature of the disease of the stomach.

The paroxysmal pain associated with the *passage of gall-stones* may sometimes be a cause of some difficulty in the diagnosis, particularly as it is often associated with tenderness in the right hypochondrium.

The chief points which distinguish this affection are its sudden invasion, its violence while it continues, the persistency of the vomiting, the co-existence of some enlargement of the liver and of an icteric tint of skin, the absence of hæmatemesis, and the immunity from epigastric tenderness, and from dyspeptic disturbances in the intervals.

The diagnosis of the site of the ulcer is sometimes aided by the effects of position in the relief of pain,[3] and in the relative rapidity with which this symptom appears after the ingestion of food, which occurs in some cases earlier when the ulcer is seated near the cardia or in the fundus than when it is situated in the pylorus or duodenum. Pain in the back has been observed to be more severe when the ulcer is on the posterior surface. Excessive tenderness on epigastric pressure has been found associated with those situated on the anterior wall. Absolute accuracy of diagnosis of the site of a duodenal[1] ulcer from one situated in the pylorus, except in cases when the former gives rise to jaundice from obstruction of the common bile duct, appears to be practically almost impossible.

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[1] Abercrombie, loc. cit. 57. Henoch, iii. 122.

[2] See a case by Empis, De la Granulie, p. 154, when this combination occurred in a case of tubercular meningitis.

[3] In some cases by Osborne, loc. cit., the patient could lie on the affected side when the stomach was empty, but this position caused pain when food had been taken.

The diagnosis of the event of *perforation* is one of extreme importance in relation to treatment. Unfortunately, the distinction of its early stages from attacks of colic is a question of great difficulty, as is attested by more than one recorded error in diagnosis.

When the event has been preceded by recognisable symptoms of ulcer, its characters can seldom be mistaken; but its sudden invasion, when the disorder has run a latent course, may easily be misapprehended. The chief criteria are the greater severity of the pain, and also of the collapse in cases of perforation, together with the early accession of general tenderness of the abdomen, with vomiting, and with other signs of peritonitis. Severer attacks of colic are often preceded by a history of flatulence and constipation, and by previous slighter forms of the disorder. They are also generally of more gradual invasion than is observed in the pain of perforation. Local spots of tenderness in the course of the intestines can usually also be discovered; and in their neighbourhood some variations in the percussion note are generally to be observed. In cases of perforation, on the other hand, the whole abdomen early becomes tympanitic. The difficulty of diagnosis should always induce caution in administering purgatives in cases where any doubt exists regarding the nature of the affection.

It must be recollected that pain after food may remain after cicatrization has been effected. It has been supposed under these circumstances to be due to the irregular contractions of the organ giving rise at times to spasm.

The diagnosis of perforation of the pleural cavity or of the lung must depend on the occurrence of the physical signs of pleurisy, or of the formation of cavities in the pulmonary tissue. That of perforation of the colon has been in some cases disclosed by the passage of undigested food, or by fæcal vomiting.

The diagnosis of ulcer from cancer of the stomach will be considered under the head of the latter disorder.

Treatment.—The principles to be followed in the treatment of this affection may be briefly summarized under the following heads:—(1)

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[1] It is desirable to recall the rapidity with which food passes from the stomach, as showing that very little reliance can be placed on any distinction of the period at which the pain occurs.

Rest. (2) The cure of conditions of the stomach which cause undue acidity from fermentation or hypersecretion. (3) The relief of pain. (4) The relief of vomiting. (5) The arrest of hæmorrhage. (6) The relief of constipation. (7) The treatment of perforation.

The measures indicated under the first two divisions are, in great part, regimenal and dietetic. Medicinal remedies also aid these, and are applicable to most of those subsequently named.

(1) It has been seen that many of the most urgent symptoms result from the movements of the stomach in the act of digestion; and our first indication is to reduce these, as far as possible, to a minimum amount, and to maintain the strength of the patient by the smallest quantity of the most digestible food necessary for this purpose, and especially to avoid distension of the stomach by any single large meal.[1]

The same principle should be kept in mind by endeavouring to reduce, as far as possible, all waste of tissue by bodily exertion; and for this purpose complete rest should be enjoined, and the warmth of the body should be fully maintained by external clothing. Confinement to bed during all the severer exacerbations is almost indispensable.

Cruveilhier's method of restricting the patient to a milk diet has been justified by the success which usually attends this plan. The milk should be given in small quantities, rarely exceeding a teacupful, at intervals of two hours; and in severer cases, or when vomiting is frequent, the amount must be restricted to table, dessert, or even teaspoonfuls. Long fasting is highly undesirable, and it is therefore better that the patient should be occasionally awakened in the night than that many hours should elapse without taking nourishment. The milk is often better borne when mixed with well-boiled arrowroot or biscuit powder, since its coagulation into masses in the stomach is thereby prevented. It should not be taken too hot; but there are great differences in individual patients with respect to the temperature at which their food can be taken. Some German authorities recommend buttermilk as a substitute, when milk in its ordinary form appears to disagree; or under these circumstances it may be diluted with water, lime-water, Carrara-water, or soda-water. The last combination is often the most agreeable to the patient. This method may often be continued for many days, or even a fortnight or three weeks, with great benefit; though at the end of this period the patients often acquire a great disrelish and even aversion to the milk diet, and some change may become necessary. It must, however, be borne in mind that occasionally an idiosyncrasy appears to exist against milk, which is not digested, but gives rise to flatulence, acidity, increased pain, and even to vomiting. In the case also of elderly people milk sometimes fails to nourish, and, unless a different diet be adopted, the emaciation and loss of strength of the patient will increase. Under such circumstances recourse must be had to animal broths, made strong, but given cool, and in similarly small quantities at each meal. If pain be severe, or vomiting urgent, I have found great benefit result from the adoption of the method proposed by Dr. Balthazar Forster,[1] of withholding all nutriment by the mouth and administering food entirely by enemata of strong beef-tea, or milk, with which brandy may be combined or not, in doses proportioned to the strength of the patient. Opium may also be given in these enemata, and it not only enables the rectum to retain them longer, but also alleviates the other symptoms. As the state of the patient improves, and the more urgent symptoms subside, more latitude may be permitted; but great caution should be exercised in this respect, even during periods extending over some years. Indigestible food of all kinds must be strictly forbidden, and great care must be continuously exercised to avoid undue distension of the stomach with any single meal, and the more so as the excessive appetite may often tempt the patient unduly to indulge in this respect. Hence, with the precaution that each meal should be small, food should be given at intervals of three or four hours, and milk may with advantage form a considerable proportion of the diet. Lightly-boiled eggs, when these agree, and the more digestible meats, which at first should be well stewed, may be cautiously indulged in. Bread should be eaten stale or toasted; but the use of vegetables should at first be restricted to potatoes in small quantities, and these are often replaced with advantage by maccaroni. The importance of a perfect mastication of the farinaceous articles of diet, and indeed of all the food, should be strongly insisted upon. Hot liquids, and especially tea and coffee, should be almost permanently excluded, and should, when possible, be replaced by milk and water, or by cocoa made from the nibs. Malt liquors are generally found to cause flatulence, and to aggravate the pain; Dr. Brinton has, however, observed, that they sometimes agree in the case of elderly people. When stimulants appear to be required, dry sherry or pale brandy, in small quantities, and largely diluted with water, is the best that can be taken.

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[1] "Verum dum consolidandum est tale ulcus, caveri debet, ne a copiosis ingestis ventriculus distendatur, detraherentur enim denuo illa quæ coire inceperant.—Præcipuum est ut nihil in victu exhibeatur quod exasperare possit hæc mala; jura carnium cum oryza cocta vel mollissimis oleribus hic sunt præcipua; vitelli ovorum, cremores hordei, avenæ ac similia parca copia simul data: ab his enim magnum solamen in doloribus illis chronicis circa ventriculum solent percipi."—Van Swieten's Comm. in Aph. Boerhaave, Ed. 1753, vol. iii. pp. 152, 153.

Sugar, since the objection made to its use by Cruveilhier, has fallen into general disrepute, and it should certainly be only moderately indulged in. It is a powerful stimulant to the mucous membrane of the stomach,[2] and it also easily undergoes acid fermentation—properties which tend to render its use undesirable.

(2, 3) The therapeutic measures under the second and third divisions include most of the remedies ordinarily employed in the treatment of ulcers of the stomach, and it is a question how far their action is directly excited on the disease itself, or in what measure their beneficial agency is due to their influence on the surrounding mucous membrane. The main object in the latter direction is to diminish hyperæmia and its causes, and to check catarrhal action; but as these indications can scarcely be distinguished separately, it will be best to speak of individual remedies which may be employed for these purposes.

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[1] Lancet, April 25, 1868, vol. i.

[2] See Blondlot, Exp. sur la Digestion, p. 223.

The influence of bismuth in this disorder is too well attested to need any apology for placing it among the first on the list of appropriate remedies. Its beneficial effects in catarrhal conditions of the mucous membrane have been already treated of. Whether or not it exerts any direct influence on the ulcerated surface may be open to question, but such an action would at the least not appear improbable. It appears also to exercise an influence in checking hypersecretion, and for this purpose may be advantageously combined with kino and opium, both of which possess a similar power, or with opium or morphia alone, when this symptom is less marked. It may be administered in the form either of the subnitrate or subcarbonate suspended in mucilage. I have rarely found it necessary to administer a larger quantity than ten grains for a dose, repeated four times in the twenty-four hours; but Dr. Brinton has employed larger quantities, as a scruple.

Opium is the remedy chiefly to be relied on for the relief of the pain and vomiting. The amount given should be sufficient for the more or less complete removal of the pain, and in severe cases several grains of the crude drug may often be given advantageously, in divided doses, in the course of twenty-four hours.

The utility of the salts of silver has been warmly disputed. There can be little probability that the nitrate in the small doses in which it can be administered (which should rarely, if ever, exceed half a grain to a grain) exercises an action on the surface of the ulcer similar to that which follows its application to external parts, since so large a proportion must, from the mucus present in the stomach, be immediately converted into the insoluble chloride. Its agency, however, in catarrhal affections of the stomach, and in cases where, from the pain and vomiting, there may have been great reason to suspect the presence of an ulcer, is so unquestioned, that, though standing second to bismuth as a remedy in this disease, it may be regarded as a valuable adjuvant to our resources in cases when this remedy does not appear to exercise its wonted beneficial effects, and it will sometimes be found to relieve pain after bismuth has failed.

The employment of alkalies, among which may be included the bicarbonates of potash, soda, and magnesia, and lime-water, should be restricted to those cases where, together with flatulency, there is evidence of acidity, resulting from fermentation in the food. The presence of free acid of this nature must exercise an injurious influence, both on the surface of the ulcer and on the mucous membrane of the stomach, which may be appropriately neutralized by these remedies given between meals. Under other circumstances their use is injurious, as tending, when given on an empty stomach, to excite the secretion of the gastric juice; and except as simple palliatives, they are of no value in the acidity resulting from hypersecretion, which is best controlled by the treatment before mentioned. In cases, however, where they are applicable, the use of the natural or artificial Carlsbad-water has been found advantageous, and it has been highly praised by Ziemssen for its aperient action. Ziemssen considers that, in addition to the neutralization which it effects on the acid contents of the stomach, it has the further beneficial action that it tends to promote peristaltic action of the viscus, and thus to prevent undue delay of the food in its interior and consequent fermentative processes.[1]

When the severer symptoms have subsided, if there be evidence of anæmia, the use of iron may be most advantageously resorted to. The neutral preparations—such as the ferrum redactum, the ferri ammon. cit., or potass tartrate, the carbonate of iron, or the mist. ferri co.—are those most suitable; they should at first be given in small doses after food, and their employment is to be discontinued if pain supervene. The recommendation of Abercrombie of the ferri sulph. in combination with aloes has been endorsed by Henoch; but I confess that I regard those above mentioned as safer remedies in these cases.

Pain of a severe kind, indicating the extension of the ulcer, requires additional care in restriction of the diet, and in enforcing absolute rest. It is, as has been already stated, most effectually relieved by opium, to which, for this purpose, hydrocyanic acid is decidedly inferior. The effect of position should also be tried. Warm cataplasms and fomentations also afford relief, and a marked effect of this kind is sometimes produced by the application of a few leeches over the epigastrium, especially if the pain is localized in this region. Their number should, however, be limited to two or three, and it is not necessary or desirable to encourage free bleeding.[2]

Counter-irritation does not seem desirable during the attacks of severe pain, and, in some instances, when an ulcer has appeared to have formed adhesions near the surface, the application of a blister has been followed by increased suffering; but in the intervals and during the course of the disease the use of remedies of this class has been recommended by many careful observers, and when combined with other suitable measures appears to have conduced to a cure.[3] Osborne recommended an issue made with caustic lime, but the least distressing counter-irritants are either mustard poultices, small blisters not exceeding the size of a five-shilling piece, or friction with croton oil.

(4) Pain is also frequently alleviated by the warm bath, and the prolonged use of this remedy has been recommended both by Cruveilhier

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[1] Sammlung Clinischer Vorträge (Volkmann), No. xv.

[2] Dr. Brinton opposed the application of leeches altogether; but the benefit that often accrues from their use, by the relief of pain, appears to counterbalance the small loss of blood which they occasion. The cases for their employment require, however, to be judiciously selected.

[3] See the case of Beclard, before quoted.

and by Andral, not only for this purpose, but also as an aid in checking vomiting. The pain of flatulent distension and spasm, though often relieved by opiates, requires occasionally, from its severity, a departure from the general principles of treatment observed in these cases. Emetics cannot be too strongly forbidden,[1] but the use of warm liquids sometimes relieves the spasm and promotes the evacuation of the flatus, or even the regurgitation of the contents of the stomach, in which these attacks commonly end. The aromatic spirit of ammonia may also be used for the same purpose. Nausea and vomiting may be treated, in addition to the remedies before quoted, by ice in small quantities, and by effervescents containing hydrocyanic acid, though the latter are of less efficacy than preparations of opium. This symptom, when obstinate, requires the most extreme restriction of the diet; and it is often advantageous during some hours, or even some days, to avoid introducing any food into the stomach, and to maintain the strength of the patient by nutrient enemata given in as small a bulk as possible. Milk, beef-tea, eggs, and, if necessary, small quantities of brandy, may be given in this manner; and Dr. Brinton, on the advice of Dr. Hawkins, employed cod-liver oil for this purpose with beneficial results. Even opiates may thus be administered with advantage, when rejected if given by the mouth. Thirst may be quenched by slowly sucking small pieces of ice.

Vomiting appears in some cases to be maintained by a loaded condition of the bowel, and in these the administration of a purgative enema is sometimes beneficial.

(5) Hæmorrhage must be controlled by cold and by direct astringents. I have found none so dependable as the acetate of lead given in doses of three or four grains, in combination with a quarter of a grain of opium, every two or three hours. Turpentine has been recommended by Hunter, and its utility has been confirmed by Drs. Graves and Seymour. Dr. Budd thinks it more useful in cases of capillary hæmorrhage than when the blood proceeds from larger vessels. The use of other remedies of this class will be further alluded to. (See Hæmorrhage.)

Ice internally may be employed with advantage for the same purpose, and this agent and also opiates are useful in checking the movements of the stomach in the effort of vomiting, by which the tendency to bleeding is necessarily aggravated. During the continuance of this symptom the most absolute rest of body must be enjoined. The diet should be restricted, in the same manner as when vomiting is present. When severe collapse is threatened, stimulant enemata may be given, and ether inhaled. It is important also to watch the fæcal evacuations after the hæmatemesis has ceased.

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[1] The danger of these was recognised by Schmidtmann, Summa Observationum Prax. Med. iii. 224, 395, who in addition to cases within his own experience, where they were followed by a fatal issue, quotes a case from Boerhaave, that of Admiral Wassenaer (Op. Omnia, 1738, p. 98), who died of rupture of the œsophagus from this cause.

(6) Constipation is always to be treated with caution; an almost universal consent has proscribed mercurial preparations as injurious. When it does not nauseate or cause vomiting, there is no better laxative than castor oil; but, in the numerous instances in which its use is prevented by the intolerance of the patient, the best substitutes are aloetics and the pil. colocynthidis composita. The administration of purgatives by the mouth should, however, as far as possible, be avoided, and the action of the bowels assisted by cold or tepid enemata, in which manner also castor oil may often be beneficially employed.

(7) When perforation is threatening or has occurred, the most absolute repose to the patient and also for the stomach is an object of primary importance. In the former case Miquel has recommended that such a position should be maintained as to leave the ulcer free from contact with the contents of the stomach. When the event has taken place, no agent appears to have any curative influence but opium, and its use must be continued for many days; the nutrition must be also conducted entirely by enemata. The only favourable recorded terminations to this event are those where these plans were pursued. If life should fortunately be prolonged, the importance of a long-restricted diet, so as to avoid the distension of the stomach, cannot be too strongly insisted upon. Finally, patients should be warned that in intervals of comparative immunity from urgent symptoms they are still in danger of relapses; and a case by Cruveilhier,[1] in which a return of the ulceration after many years of immunity followed the free exhibition of purgatives for a cerebral affection, may well awaken the question put by that distinguished author, whether these remedies were not truly the cause of the relapse, and should induce caution in all treatment, hygienic and medicinal, of patients in whom the disorder has once existed. The later effects arising from constriction of the pyloric orifice may be obviated by the use of a diet chiefly solid in order to avoid distension by flatulence, or, when this has occurred, by the employment of the stomach pump after Kussmaul's method.[2]

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[1] Path. Anat., Liv. xx. p. 2.

[2] See p. 985; also Ziemssen, loc. cit.

VII.—Cancer of the Stomach.

Definition.—A disease of uncertain duration, characterised by the symptoms of pain, vomiting, perversions of the secretions of the stomach, and disturbances of the appetite and digestion, and tending to a fatal termination by marasmus, cachexia, or hæmorrhage; whose essential anatomical character depends on the development in the coats of the organ of a heterologous growth presenting the structural peculiarities and pathologial course of cancer.

History.—Tumours forming in the coats of the stomach, and tending to ulcerate, have been known from very early times. Their nature, however, has only become more strictly defined by the extension of the knowledge of pathological anatomy, which has revealed the dependence of many cases formerly described as melæna and cardialgia upon growths of this nature.[1]

Etiology.—*Frequency.*—The estimates of the comparative frequency of this disease in relation to all other causes of death vary between 0·6 and 2½ per cent.[2] It appears, therefore, that the disease is less common than the simple ulcer of the stomach, but its extreme fatality places it on about the same level in respect to its frequency as a cause of death.

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[1] The most complete accounts of the pathological anatomy of this disease are to be found in Carswell, Illustrations of the Elementary Forms of Disease; Rokitanski, Path. Anat.; and Cruveilhier, Path. Anat. (excellent illustrations of the colloid form). Their clinical history, etiology, and pathological anatomy have also more lately been described by Walshe, On Cancer; Lebert, Traité des Malad. Cancereuses; Brinton, Med.-Chir. Rev. 1857, and Diseases of the Stomach; Dittrich, Prag. Viertel-Jahresch. vol. xvii. For other illustrations and cases see Abercrombie, loc. cit.; Réné, Prus. Rech. Nouvelles sur la Nature et le Traitement du Cancer de l'Estomac; Barras, Préc. Anat. sur le Cancer de l'Estomac; Müller, Krankhaften Geschwülste; Bruch, Henle and Pfeuffer's Zeitsch. 1849; Bennett, Cancerous and Canceroid Growths and Clinical Medicine; Köhler, Krebs und Scheinkrebs, 1853; Broca, Mem. Acad. de Méd. 1852. See also articles in Dict. des Sciences Méd. and Dict. de Méd. by Bayle, and Fayol, and Ferus; Chardel, Dég. Squirrheuses de l'Estomac, 1804; Valleix, Guide du Méd. Pract; Handfield Jones, Budd, and Habershon, before quoted. For other references see Walshe and Lebert, and articles in Cyc. Pract. Med. and Copland's Dictionary.

[2] The smallest number is given by Tanchou (Rech. sur le Traitement Médical des Tumeurs du Sein, Paris, 1844), founded on an aggregate of 382,851 deaths in Paris, during the years 1830–40. Virchow (Verhand. Phys.-Med. Gesell., Würzburg, vol. x. and Krankhaften Geschwülste, vol. i.) and Brinton (loc. cit.) agree in their estimates of about 1 per cent.: that of the former author being based on a total of 3,390 deaths in the mortality returns of Würzburg during the years 1852-5; that of the latter on a collection of records of 8,468 post-mortem examinations in various London hospitals. An almost similar correspondence in an average of 2 to 2½ per cent. is attained by Marc d'Espine (Statistique mortuaire du Canton de Genève pendant les années 1838-1855, Echo Médicale, 1858, quoted by Lebert and Virchow,) and by Willigk (Prager Viertel-Jahresch. Nos. 38, 44, 50, 51); that of the former being derived from the registers of the Canton of Geneva, while that of the latter is founded on the recorded necropsies, amounting to 6,196 cases, in the hospitals at Prague. Willigk's percentage of the relative frequency of cancer of the stomach to other cancers agrees very closely with Virchow's.

Regarded in relation to primary cancers in general, that of the stomach is one of the most common occurrence. Tanchou's tables represent it as forming 25·2 per cent., Marc d'Espine's 45 per cent., and Virchow's 34·9 per cent. of the whole number of cancers recorded, equalling, if not exceeding, in frequency those of the uterus and mamma.

Age.—Dr. Brinton's returns from 600 cases show that three-fourths of these occur between 40 and 70; and that the greatest number is met with between 50 and 60; though, allowing for the number of all persons living, the maximum liability is found between 60 and 70—a result very closely corresponding for the particular organ with that deduced by Dr. Walshe for the whole class of these diseases.[1] It is decidedly a rare event in the earlier periods of life, but a case of congenital scirrhus of the stomach is recorded by Wilkinson;[2] though ordinarily, when occurring in this organ during childhood, it tends to assume the medullary form.

Sex.—The data on this head are somewhat uncertain. Brinton, from 784 cases, considered that there was an excess in the male sex in the frequency of its appearance. As other writers have, however, given very different proportions,[3] the sexual influence cannot be considered very strong in determining or preventing the localization of the disease in the stomach; nor does the united influence of age and sex present at all the same remarkable etiological features as are observed in the case of gastric ulcer.

The remaining causes of the disease in this special seat are as obscure as those which determine its appearance in other parts.

Hereditary transmission appears to operate with the same uncertainty as prevails with regard to the whole class of Tumours. Its influence as an occasional predisposing cause can scarcely be questioned, and the case of the Napoleon family, so often cited, is a striking illustration of this tendency.

The influence of direct irritation[4] or inflammatory action main-

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[1] Loc. cit. pp. 149, 151.

[2] Edin. Med. Journ. Jan. 1841, quoted by Dr. Walshe, p. 146.

[3]

Brinton	gives	784 cases;	440 males,		344 females.	
Louis	,,	33 ,,	20	,,	13	,,
Lebert	,,	42 ,,	19	,,	23	,,
Marc d'Espine	,,	116 ,,	54	,,	62	,,
Dittrich	,,	159 ,,	64	,,	95	,,
Willigk	,,	169 ,,	83	,,	86	,,

In Dittrich's and Willigk's cases there was an excess in the number of females dying in the hospital. Brinton considered that the excess in the male sex was due to the greater proclivity of the generative organs in the female to become the seat of cancer.

[4] Among the curiosities of this class may be mentioned cases where corrosive poisons have been followed by an outbreak of cancer: as after nitric acid, recorded by Andral, Clin. Méd. ii. 99; or arsenic, Dittrich, Prag. Viertel-Jahreschrift, xix. pp. 110, 114. The latter case may, however, be well contrasted with one quoted by Dr. Walshe, when a mass of arsenic was encapsuled in the stomach, without further apparent injury. (Cancer, p. 167.)

tained by Boerhaave and Van Swieten,[1] and also by Broussais and Andral,[2] and by other writers of the French school, can only have a direct influence attributable to them under circumstances of some[3] as yet unknown constitutional predisposition. A direct effect also can scarcely be attributed to habits of spirit-drinking, or to blows, or other mechanical injuries or pressure on the epigastric region; nor even to depressing emotions when independent of the unknown antecedent conditions, though this class of causes has frequently been observed to favour either the immediate outbreak or the more active development of the growth.

What the local predisposition may be that causes the stomach with such great proportionate frequency to become the seat of this disease can only be in great measure a matter for conjecture. Virchow's argument that the organs and parts of organs which most frequently suffer from morbid growths are either those which, from their position or structure, are most exposed to or susceptible of injurious influences from external agencies, or are those whose nutritive processes[4] are conducted under special and peculiar conditions, finds at least a basis in the case of cancer of the stomach: but it is also deserving of notice, as pointed out by Dr. Brinton in relation to this question, that the disease only very rarely commences on the surface of the mucous membrane.

Marc d'Espine's returns show an excess of mortality from cancer of the stomach in the wealthier classes. Of twenty-one cases there were fifteen rich to six poor,[5] but these numbers appear too small to serve as the foundation of a comprehensive induction.

Bamberger states that it is most common in flabby and fat persons, but gives no further detail.

SYMPTOMS.—Cases of cancer of the stomach may be divided into two distinct groups,—one in which the disease, even when attended by extensive ulceration, may run an almost latent course, and even arrive at a fatal termination, unmarked by any severer symptoms than anorexia and dyspeptic disturbances of a comparatively insignificant character, but attended by a gradually increasing emaciation and loss of strength—and a second, when its presence excites the more formidable disturbances of pain, vomiting, and hæmorrhage. A third variety, however, occasionally occurs, in which cases that have presented the features of the first-named class may towards the close assume the more distressing characters of the second. A fourth also is sometimes met with, where the pain or vomiting, which have been the first symptoms, has ceased, and the disease has advanced to a fatal termination, and where the only distinct symptom has been a steadily progressing emaciation (Abercrombie).

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[1] Comm. in Aph. Boerhaave, 1758, vol. iii. pp. 147, 150.

[2] Clin. Méd. ii. 31, 60, 61.

[3] Symptoms of chronic inflammatory action or dyspeptic disturbance have indeed been noticed to precede for many years the severer symptoms; but it is doubtful whether, on the one hand, these may have not been caused by a latent growth of the disease, and, on the other, how far, considering the prevalence of these symptoms in patients who do not become cancerous, any influence in the production of the disorder can be ascribed to such derangements. Beau, Gaz. de Hôpitaux, 1859, pp. 390-1, says that an "idiopathic" dyspepsia may long precede cancer of the stomach. Lebert's analysis, however, would show that in the majority of cases of cancer of the stomach the digestion has been accomplished naturally up to the period of the appearance of the disease.

[4] Dr. Walshe's critical remarks on this question deserve the most attentive consideration, from the remarkable logical acumen with which he has handled it, loc. cit. pp. 94, 95.

[5] Ann. d'Hygiène, 1847, xxxvii. p. 323, quoted by Lebert.

Cases of the first kind, though by no means rare in the history of medicine, do not form a large proportion of the whole.[1] The absence of the more prominent symptoms may often be traced to conditions affecting either the site of the growth, the rate of its enlargement, or the depth and extent to which ulceration has proceeded, or the relation of the nerves or vessels of the stomach to the tumour.

In its ordinary course the disease is usually first manifested by symptoms of dyspepsia, beginning without apparent cause in a person who has arrived at middle age, and who often has previously enjoyed good health. These may be at first very undefined, consisting of weight and uneasiness felt at the epigastrium after taking food, and followed by gaseous, acid, or insipid eructations. The tongue in the meantime is usually found unaffected, pale, or presenting its normal appearance. Thirst is generally absent. Loss of appetite is often an early and prominent feature. The strength fails, and signs of emaciation, at first slight, become increasingly distinct. As the disease advances the epigastric uneasiness passes into pain, which is often of considerable severity. Vomiting also takes place at irregular intervals. Rare at first, and attended only with the rejection of food, or of mucus, which may be stained or mottled with streaks, specks, or flakes of rusty, sooty, or coffee-ground tint, it occurs more frequently as the disorder progresses. Flatulence and constipation become at this period prominent symptoms, and the patient is often dejected, morose, irritable, or desponding. At variable periods in the history of the case a tumour becomes perceptible in the epigastric region. In proportion as the disease is more fully declared, the epigastric pain grows severe and distressing. Vomiting is frequent, and large quantities of more or less altered blood are at times ejected. The emaciation deepens into cachexia; the skin acquires an earthy tint; diarrhœa alternates with constipation; febrile action, akin to hectic, sometimes appears towards the close, though usually this symptom is absent; dropsy, general or local, occasionally supervenes; and the patient dies exhausted after one or two years of suffering, and with an extreme degree of marasmus.

These symptoms, however, often differ considerably in their relative intensity, and also in the order of their occurrence, and appear to merit some separate consideration.

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[1] For observations of this nature see Pemberton, Dis. of Abdominal Viscera, p. 84; Sir T. Watson, Princ. and Pract. Physic, vol. ii.; Seymour, Med.-Chir. Trans. vol. xiv.; Andral, Clin. Méd. ii. Cruveilhier also gives a case where the appetite and digestion were preserved, and the only symptoms were dropsy, emaciation, and pyrexia: Path. Anat., Liv. x. A very similar one is recorded by Abercrombie. Even extensive ulceration appears in some cases to have been attended by little or no vomiting, and by only comparatively slight disturbance of the digestion.

The duration of the preliminary stage is very variable, and the disturbances in the digestion may present nothing characteristic, and may even be entirely absent up to the fatal termination (Cruveilhier).

Anorexia, however, though by no means constant (as the appetite is in some cases maintained to the close of life), is a very distinctive feature of the disorder. Generally it proceeds *pari passu* with the pain and with the increasing cachexia and debility, to which, in many cases, it stands in direct relationship. Sometimes, however, it occurs early in the disease, and appears to be quite unconnected with the latter symptom, and under these circumstances it is, according to Brinton,[1] more marked in proportion to the youth of the patient and to the softness of the cancerous growth. There are, however, intermissions sometimes of considerable duration, in this disrelish for food, and, instead of being lost, the appetite may be capricious or fanciful. According to Lebert's and Brinton's estimates, anorexia occurs sooner or later in from 78 to 85 per cent. of all cases of cancer of the stomach, and thus forms a remarkable contrast with the condition of the appetite in cases of gastric ulcer. When, however, vomiting is present, and especially when this arises from obstruction of the pylorus, the appetite may be found to be increased.

Pain is the most constant,[2] as well as the most marked, symptom. It is frequently the first in its appearance, and is often during a long period the only evidence of the disease. At first it seldom presents the same intensity as in the later stages of the disorder, and it may then only exist as a dull aching felt in the epigastric region, or in the back.[3] It is, however, often of intense severity, and is described as burning, tearing, or lancinating;[4] but this latter character is not always observed. There are, not unfrequently, complete intermissions in its appearance, which may last over many days, or even weeks; in other instances, even when the pain is of great severity, it may be nearly continuous. In some cases it may occur in paroxysms of considerable severity, resembling attacks of cardialgia, or even colic, obliging the patient to double himself up for many hours, and it may occur in this manner, unattended by other symptoms, for years.[5] These attacks are not, however, so frequent in cancer as in ulcer of the stomach, and the character and intensity of the pain are influenced by the occurrence of ulceration, by the invasion of large branches of nerves,[1] by the position of the ulcerated tumour, and by the extent of surface affected.

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[1] This author records a case where the anorexia was only manifested by a sudden disrelish for tobacco in an habitual smoker, but which, combined with a cachectic appearance, induced both the patient's attendant and himself to diagnose the existence of cancer, which was shortly after verified.

[2] Lebert says that it occurs in five-sixths, and Brinton in 92 per cent. of the cases which they have collected.

[3] As in a case by Sir T. Watson, where the pain was obscurely seated in the lumbar region, giving rise to the suspicion of renal calculus, but also resembling lumbago.

[4] Authorities are divided in their statements regarding this character of the pain. Bamberger, Lebert, and Brinton speak of it as being frequent; Walshe, Andral, Henoch, though affirming its occasional occurrence, deny its comparative frequency.

[5] The case of the first Napoleon is an instance of this. One presenting very similar characters has come under my own observation.

In site it may either be localized, or it may extend through the whole epigastric region. It is often felt in the back behind the scapulæ and in the course of the spine.[2] It is sometimes, especially when a tumour can be felt, associated with distinct tenderness on pressure over the site of the growth, which is more marked in proportion to the superficial character of the latter, though this symptom is not unfrequently absent.

The pain is commonly, at least in the later stages, aggravated by the ingestion of food; but in the earlier periods of the disease this relation is not so distinctly observed as in cases of ulceration, nor is the pain so commonly relieved by vomiting.

Vomiting, though less constant, and usually appearing later than pain, is, however, a symptom of great frequency, being recorded, according to Brinton, in 87½ per cent. of all cases.

It is most frequent when there is ulceration of the surface, or obstruction of the orifices by the tumour; but that it does not depend exclusively on either of these conditions is shown by its absence in some cases where they have both been found, and by its presence in others in early stages of this disease, and when neither exist.[3] Its appearance under the latter circumstances is probably due to the direct irritation of the excito-motor nerves of the organ by the progress of the growth, and to the same influence must be attributed its occurrence in those cases when it has been excited by pressure on the tumour (Lebert).

The period at which it follows eating is generally influenced by the position of the cancer. When the cardia is obstructed, the food is usually rejected immediately after deglutition, unless the return is delayed by dilatation of the œsophagus. When the pylorus is the seat of the disease, food may be retained for some time before it is returned.[4]

Occasionally, however, vomiting occurs when the stomach is empty, and this is sometimes observed in the morning, when mucus is usually brought up—a symptom possibly referable to co-existent gastric catarrh.

Its frequency usually increases with the progress of the disease, but long intermissions between the attacks are common in all stages. The intervals also tend to become longer when dilatation of the stomach has ensued in consequence of obstruction of the pylorus; but when vomiting occurs under these circumstances, enormous quantities of acid and fermenting food are rejected from the stomach.

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[1] As in a case by Sir T. Watson.

[2] Brinton says that he has observed scapular pain in cases of cancer of the cardia, and pain in the lower dorsal and lumbar region, when the growth has been found on the posterior surface of the stomach.

[3] Lebert. See also a case by Dittrich, loc. cit. p. 114, where there was almost complete scirrhous degeneration of the coats of the whole organ, and yet vomiting was absent. I have met with a similar case.

[4] The act of vomiting does not appear to be determined by the position of the tumour, independently of the obstructions to which it may give rise. See Lebert, p. 505.

In some cases vomiting, which has been frequent in the earlier stages, may almost entirely cease towards its close,—a change which has been occasionally traced to the pyloric orifice again becoming opened by ulceration.

The matters vomited vary. Mucus has already been alluded to, and the ejection of an acid fluid apparently derived from hypersecretion is not very uncommon.[1] When there is an obstruction at the cardia, the food is returned but little altered, and merely macerated and covered with a layer of mucus. When the pylorus is obstructed, food undergoes the changes before noticed, and sarcinæ and torulæ are often found in the yeasty scum which forms on the surface. In fact, though sarcinæ occur in the matters vomited in a great number of stomach affections, their presence is more frequently observed in cases of cancer of the pylorus than in any other single disease to which the organ is liable. Cancer cells, also, are said to have been met with in the ejected matters, but the cases in which their distinctive features can be recognised must be extremely rare.[2] When sloughing action is taking place in the cancer, the vomited matters and the eructations may be offensive, but this is in some measure prevented by the antiseptic action of the gastric juice.

Hiccough sometimes forms a very distressing symptom, which, however, usually only appears with any severity towards the close of these cases.[3]

Hæmorrhage to a greater or less degree occurs, according to the statements of Lebert and Brinton, in nearly one-half of the recorded cases of this disease. It may be divided into two classes: slight, when only small amounts of rusty, sooty, or chocolate-tinted specks appear in the matters vomited; and larger losses of blood, in which pints may be ejected.

The former class is more frequent in cancer than in ulcer, since in addition to the congested condition of the mucous membrane surrounding the tumour, in which varicose veins have sometimes been observed (Andral), the capillary vessels on the surface of the tumours are liable to bleed, a tendency especially noticed in fungating excrescences.[4]

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[1] Golding Bird states that in a case of scirrhus of the pylorus, where the patient vomited several pints of fluid in the twenty-four hours, he found a "quantity of free hydrochloric acid, equal in each pint to 22 grammes of pharmaceutical acid, in addition to a considerable quantity of some organic acid (lactic ?), sufficient to neutralize nearly 7 grains of pure potash; at another time the hydrochloric acid nearly disappeared, and the quantities of organic acid in each pint required for saturation nearly 17 grains of the alkali." (Urinary Deposits, 1857, p. 162.)

[2] See *ante*.

[3] Bamberger has observed that it is not caused by implication of the diaphragm, but is more often connected with extension of the growth to the peritoneal surface of the stomach.

[4] Hence these rusty coffee-ground vomitings, though common to both disorders, were regarded before the time of Cruveilhier (who showed that they also took place in ulcer) as peculiarly characteristic of cancer of the stomach.

This form usually appears in the early stages of the disorder, while the larger hæmorrhages are usually associated with rapid sloughing destructive processes, and, in contrast to the other variety, they are less frequent in cancerous than in simple ulcerative disease. The symptoms of the larger hæmorrhages are similar to those observed during the progress of simple ulcer; and in these also the blood may escape by stool, and may give rise to melæna and diarrhœa.

The discovery of a tumour forms one of the most important elements in the recognition of the nature of the disease. From the returns of Brinton and Lebert, it may be felt in from 70 to 80 per cent. of all cases observed, and, even when not distinctly perceptible, there is often an induration associated with dulness of percussion note over some portion of the regions occupied by the stomach. The position of the tumour necessarily influences the facility with which it is detected by physical examination, and those in the cardia or in the posterior wall may occasionally elude observation.

The period at which it is felt is usually when other symptoms have existed for some time; but this, again, depends in great measure on the position in which it is developed. Its site is usually at the epigastric region, or (from the comparative frequency with which the pylorus is affected) in the right hypochondrium. Brinton states, that in the female sex it may be found in the umbilical region in nearly two-thirds of all cases in which it is discoverable—a peculiarity due, in a great measure, to the effects of the compression of the lower part of the thorax by stays. Similar displacements, common to both sexes, may be due to the weight of the tumour dragging down the pyloric end of the stomach, when this is not retained by adhesions in its original site. The tumour feels hard and irregular to the hand, especially when it is large and situated near the anterior surface; it is generally immoveable by manipulation, but alters its position through distension of the stomach by food, or by gradually increasing dilatation of the viscus. Sometimes it may disappear entirely for many days, either from the twisting of the stomach upon its axis, by which the pylorus is brought below the liver, or from its being covered by a distended colon. In some cases, Brinton thinks that its complete disappearance may be due to sloughing and destruction of the growth. Pulsation in the tumour is not uncommonly observed, due probably, in most cases, to an impulse derived from the abdominal aorta. Instead of a circumscribed mass, the whole epigastric region may be hard, prominent, and resisting, and, in some cases, the form of portions of the stomach may become prominent through the abdominal wall[1]—a condition which usually depends on extensive infiltration of the coats of the stomach by the cancerous growth. In these cases the percussion note, instead of being absolutely dull, may have a muffled tympanitic resonance; a peculiar tinkling sound has also sometimes been heard when liquids are swallowed, arising from the fall of the fluid into the dense cavity (Bamberger).

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[1] Louis, Mem. Anat. Path. 130, quoted by Dr. Walshe.

The signs of contraction or distension in the stomach consequent upon pyloric or cardiac stenosis will be given under the head of these affections.

The tongue presents but few characteristic features. Its appearance is not necessarily affected by the cancer, and the varieties of fur, aphthæ, &c. occasionally observed, depend more on the general condition of the patient, and especially on the co-existence or absence of a catarrhal condition of the mucous membrane. An excessive salivation with characters similar to those observed in ulcer has been sometimes noticed.

The cachexia in cancer of the stomach very frequently presents the most characteristic features of this disease. It sometimes appears early in the disorder, though its progress is usually proportioned to the severity of the vomiting, hæmorrhage, pain, and disturbances of digestion. In the early stages, however, and even sometimes at an advanced period, there may be very little external evidence of disturbance of health.

When the cachexia becomes marked, the face is pale and sunken, with deepening of the naso-labial wrinkles. The expression is anxious and careworn, or indicative of pain. The skin acquires an opaque earthy tint, which is equally marked in fair as in dark-complexioned people ; in other cases, and particularly when hæmorrhage has occurred, it has the waxy look of extreme anæmia. It is often dry and rough from the desquamation of the cuticle and from the want of perspiration. Jaundice or a straw-coloured icteroid tint of skin is not uncommon. The lighter shades of jaundice are rather more frequent; the severer cases depend on pressure by the growth in the stomach on the common bile duct, or on secondary formations in the liver.

Œdema often appears towards the close of the complaint, arising either from the general hydræmia, or limited to one of the lower extremities, through venous obstruction from thrombosis. Ascites is also occasionally, but not constantly, observed; its occurrence depends either on the pressure exercised by the tumour on the portal vein, or on cancerous formations in the peritoneum, of which latter cause the most frequent examples are afforded, in proportion to their number, by the extensions of the colloid variety. Obstructions of the arterial circulation, either by thrombosis or embolism, are also occasionally observed; they then give rise to intense pain in the limb affected, with burning or cramp-like sensations, and sometimes to great temporary hyperæsthesia of the skin, which, at a later period, are followed by gangrene of the part below the seat of obstruction.

Febrile reaction is the exception rather than the rule, but it is sometimes very distinctly marked, and especially towards the close of the complaint.[1] Its occurrence is frequently due to some secondary inflammatory action, among which pneumonia, associated either with cancerous deposits in the lung, or of the hypostatic variety, is one of the most frequent causes.

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[1] I have, however, known febrile action, due apparently to an abscess formed behind the stomach, persist during nearly two months.

Emaciation and loss of strength usually progress rapidly from the first outset of the severer symptoms, though occasionally the nutrition and general vigour are maintained for a year or eighteen months (Lebert). In many cases, however, these symptoms are among the most prominent present; and there are few in which, towards the close, marasmus does not form a very marked feature; while in others, even at the commencement, and while the other symptoms are still obscure, the presence of these signs may, as has been before noticed, form valuable indications of the nature of the disease.

THE DURATION of the disease, regarded from the origin of the cancerous growth, is very indefinite; in fact, it appears impossible in all cases to fix the period of its commencement. The length of time during which some patients have suffered from even severe pain (as in the case of Napoleon, in whom this symptom occurred at intervals for nine years before his death—Abercrombie) points to the probability that the disease may occasionally persist long, without much disturbance of the general health, or of the functions of the stomach. Ordinarily this period of comparative latency rarely extends beyond two or three months, though it has been known to last for a year and a half (Lebert).

The average duration is estimated by Dr. Brinton, from 198 cases, as amounting to 12½ months, which corresponds pretty closely with the results obtained from smaller numbers by Lebert and Valleix. The minimum course which I can find recorded is one of four months (Valleix). The longest period which has been known to elapse from the first distinctive symptom to a fatal termination has been three years and a half.

PROGNOSIS.—The termination has been invariably fatal: only very untrustworthy evidence has been afforded of the cure of the growth by enucleation and cicatrization; and evidences derived from cicatrices are uncertain, as we can never exclude the possibility of their having resulted from chronic ulcer (which has been shown to be a more common disease than cancer); nor can the same probability in favour of their origin from simple ulcers be denied in those instances where a cancer and cicatrices have been found co-existent in the same stomach.

Cancer, it is known, possesses the destructive peculiarity, that it is never circumscribed, but that the tissue around its margin is constantly found presenting more or less evident perversions of development with structures analogous to those of the morbid growth. In the presence, therefore, of the direct clinical experience which the course of the disease constantly affords us, and owing to the fallacious resemblance between its symptoms and those of ulcer, the evidence of the possibility of its cure must be established by more direct proof than has hitherto been furnished, or than the circumstances attending its formation in this position appear capable of affording, before this can be admitted, even on hypothetical grounds, to alleviate in any way the gloomy prognosis to which its recognition must always give rise.

The probability of a rapid course is determined by the severity of the vomiting and hæmorrhage. Lebert considers early vomiting an unfavourable symptom. Cases exhibiting it have frequently terminated fatally in from four to six months.

PATHOLOGY.—The pathology of the disease will only be treated of in this place in relation to special anatomical peculiarities affecting the organ.

The growth occurs in all the known forms assumed by cancer in the order of frequency here enumerated: scirrhus; medullary (and combinations of these); colloid, either simple or combined with either of the above; villous; and melanoid. Of these, scirrhus is found in nearly three-fourths of the whole number,[1] but it is seldom unattended by softer portions on the surface or margins, which approximate in their structure and character to the medullary type.

The seat of the cancer is, in the majority of cases, in the pyloric region.[2] The cardia, from Brinton's and Lebert's returns, only appears to suffer in about 10 per cent. of all cases. Extensions beyond the pylorus to the duodenum are extremely rare;[3] but when the pylorus is affected, the cancer usually extends around the whole circumference of the valve, and thence invades the smaller curvature. When the cardia is affected, the growth is generally, but not constantly, found to extend into the lower part of the œsophagus. The proportion of all cases in which the orifices taken collectively are affected amounts to 71 per cent.; an excess in these parts which, as Brinton has pointed out, is considerably greater than that observed in cases of simple ulcers.

Cases where large tracts of the stomach are invaded are usually those where the cancer is of the colloid variety, but it is occasionally noticed in other forms.[4]

The growth, when of the scirrhous or medullary forms, almost invariably takes its origin in the submucous tissue. It has been found, however, by Dittrich,[5] commencing in the subserous cellular tissue.[6] The development of the colloid form will be more particularly alluded to hereafter.

The special forms are each connected with some anatomical peculiarities, which require a separate description.

Scirrhus of the stomach presents the same contracting, indurating characters that distinguish the growth in other parts. Commencing, as just stated, in the submucous tissue, it thence extends in about equal degrees into the mucous membrane, and also into the muscular coats. The invasion of the mucous membrane is frequently marked by striated radiating lines of a cicatricial appearance, attended with destruction and atrophy of the glandular textures and induration of this coat, and sometimes by retractions and depressions of portions of the, as yet, unbroken surface. The true mucous structure (viz. the glands and villi), however, often resists during a long period the cancerous encroachment, although the membrane early becomes fixed and immoveable upon the submucous tissue.

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[1] Brinton, from 180 cases.

[2] Of 360 cases Brinton found that this was affected in 60 per cent; Lebert, 34 times in 57 cases.

[3] Cases in which this has occurred have been recorded both by Lebert and by Brinton.

[4] Brinton in 360 cases has found 13 in which the whole stomach was found thus degenerated.

[5] Prager Viertel-Jahresch. xvii. p. 6.

[6] In some of these cases this author has observed a scirrhous degeneration of this layer, together with a medullary growth in the submucous tissue, the intervening muscular coat having been unaffected.

Through the character of the growth, in which as elsewhere the fibrous stroma greatly predominates over the cellular elements, the parts affected by it become converted into a firm, unyielding mass, in which all their distinctive features are lost. When large tracts are thus affected, the disease may at first sight present a great resemblance to fibrous thickening, or to hypertrophy of the coats of the organ.[1]

The invasion of the muscular tissue by the growth takes place, as pointed out by Rokitanski, in the intermuscular septa, which form meshes enclosing spaces containing unaffected muscular fibre. The portions thus included present a reddish or a semi-transparent appearance, and their histological elements are at first greatly enlarged, but subsequently degenerate into the cancer structures.

The effect of these changes is to produce great contraction of the parts in which they occur. Either at the pylorus or cardia the immediate result is an extreme narrowing of their openings, which is often heightened by irregular masses protruding at the surface, and by polypoid vegetations, which, though less common than in the medullary variety, sometimes accompany scirrhous cancer.

When large tracts of the coats are thus invaded, the stomach may be externally shrunken and contracted, so as to resemble a fowl's gizzard in appearance,[2] with dense inflexible walls, which may even attain the thickness of an inch. The small curvature may be so shortened as to bring the pylorus and cardia into close proximity, and the inner surfaces of the interior and posterior walls may be almost completely in contact.[3]

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[1] The diagnosis between these two alterations is not always an easy one, since, as in other parts affected by scirrhus, large tracts of tissue may often be found presenting nothing but a dense fibrous structure, and devoid of the cell-structures which are usually found in the more open meshes of the stroma, though these, as pointed out by Rokitanski, can usually be found at the margins of the growth. In addition to the cancerous character of the margin, the scirrhous growth is also distinguished by its affecting all coats equally, by their fusion into a uniform mass of pearly whiteness, and presenting the gristly cartilaginous texture characteristic of this form, by the immoveability, except at the earliest stages, of the mucous membrane upon the tissues beneath, and by the destruction of the normal appearances of the muscular layer. Two cases exhibiting the contrast of these forms are recorded by Dr. Wilks, Path. Soc. Trans. x. 136, xiii. 83.

[2] As in a specimen cited by Dr. Walshe.

[3] Dittrich. In one such case the mucous membrane was found smooth and shining, as if from the effects of attrition; but later it may become the seat of ulcerations hereafter to be described.

Medullary cancer, commencing in the same tissues as the scirrhous variety, appears usually in the form of nodules, in masses of varying degrees of softness, and of a cerebriform appearance; more rarely it occurs as an infiltration of the different coats.

Though the peritoneum suffers with less frequency, the mucous membrane is invaded with greater rapidity by medullary than by scirrhous cancer. The nodules, when seen on the external surface, present, with the exception of their softness and cerebriform appearance, very few peculiarities. In some cases the growth on the mucous membrane tends to form large fungating excrescences, in which an exaggeration of the villous type is observable. These constitute the varieties of the so-called villous cancer, which may sometimes form large tumours, thickly covered with the hypertrophied villi.[1]

The melanoid form is very rarely observed.[2] In some cases it appears as small scattered nodules in or under the mucous membrane, which present the ordinary characters of medullary cancer, with the exception that their cell structures are loaded with melanic pigment.

The colloid or gum cancer has its most frequent seat in the stomach, but even here it is not of comparatively frequent occurrence.[3] Its site of origin appears to be rather more doubtful than is the case with the other growths before alluded to. Some authors state that it begins in the submucous tissue, others in the subserous.[4] My own opinion, which I would however state with some hesitation, is that this variety of "cancer" is essentially of glandular origin, and akin to those forms of epithelioma of the skin described by Remak,[5] which commence with heterologous extension of the sebaceous and sudoriparous glands into the deeper structures.[6]

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[1] Their structure can be very well seen when examined under water. Each villus contains a loop of vessels, and the larger ones are usually filled with cancer cells. The delicacy of the structure of the capillary walls, which are often only covered by a single layer of epithelium, and the softness of the whole growth, together with its extreme vascularity, render this variety a dangerous source of hæmorrhage.

[2] Only three times in 180 cases, Brinton; once in 160 cases, Dittrich. In Dittrich's case it was coincident with a general dissemination of melanoid tumours throughout the body.

[3] In 180 cases of Dr. Brinton's, colloid was found 17 times. In 160 of Dittrich's it only occurred in 11 specimens, and in three only of these was it uncombined with either scirrhous or medullary growths.

[4] Brinton, loc. cit. 239.

[5] Deutsche Klinik, 1854, p. 70 et seq.

[6] I must state, that since my attention has been devoted to this question I have only had an opportunity within the past four years of examining one recent specimen, in which, however, the glandular origin was most distinct; and my observation on preparations preserved in spirit has, though not conclusive, tended to confirm this view. In the former case there was a distinct colloid mass obstructing the pylorus, unassociated with any other form of cancer in the stomach, though attended with many polypoid growths from the mucous membrane. Two points of great interest in connexion with it were the association with an ordinary epithelioma of the lower third of the œsophagus, which did not show traces of glandular origin, and which was attended by secondary epithelioma of the mediastinal lymphatics; while in the retroperitoneal lymphatic glands, which lay immediately below the diaphragm (and had probably therefore been infected directly

It is possible that, when apparently commencing in the deeper coats, this growth may, under such circumstances, take origin in the little glandular masses which occasionally are found in these parts, and having no connexion with the rest of the mucous membrane, and of which I have seen some examples.[1] However originating, this form of cancer tends to spread over large surfaces of the mucous membrane, which is thus greatly thickened, and has its normal texture converted into a reticular structure, with spaces filled with "colloid" material. Its tendency to invade deeper issues is also very considerable, and it frequently extends to the peritoneal surface, and thence to the omentum.

In their subsequent course all cancers of the stomach have certain characters in common, marked only by minor shades of variation dependent on their peculiarities of growth.

Changes in the form of the stomach from the contraction of scirrhus have been already alluded to. Diminution in the size of the organ may also occur from obstruction of the cardiac orifice. Dilatation is a very common effect of obstruction of the pyloric orifice; and it is sometimes attended with thickening, at others with thinning of the coats. Thickening when present usually predominates in the muscular layers, which then undergo a true hypertrophy. The size attained under these circumstances by the organ is sometimes such as to fill the whole abdominal cavity, and to extend even to the pubes.

Adhesions are very common to adjacent viscera when the cancer has extended through the peritoneal coat. The most common of these are to the liver, pancreas, omentum, spleen, or diaphragm, or to the abdominal wall.

Displacements, unless prevented by adhesions to neighbouring viscera, may be caused by the weight of the tumour dragging the stomach into the lower portions of the abdomen; and under these circumstances it may become fixed by new adhesions in abnormal situations, and the pyloric portion may be found in the right iliac fossa, or even in the pelvis, adhering to the intestines, uterus, ovaries, or bladder.

Ulceration is common to all varieties of cancer. It is most marked in the softer medullary forms, when it often takes on the form of sloughing,

from the stomach), I found distinct masses of a medullary character. I think it very possible that the secreting glands of the stomach may undergo this abnormal development in cases where the primary disease has been a scirrhous or medullary growth of the submucous tissue, but I doubt whether any direct *metamorphosis* can take place from either scirrhous or medullary cancer into the colloid variety. A glandular tumour of the pylorus having a structure very similar to what I observed in the case of colloid just quoted, is figured by Dr. Hughes Bennett, Clin. Lect. 1865, p. 495. Its structure is, however, described as thickened, indurated, and white.

[1] These are mentioned by Rokitanski; the largest growth of this kind which I can find recorded is by Loeschner and Lambl, Berichte aus dem Franz Joseph Kinder Spital. That glandular growths may assume all the characters of "malignant" structures is shown by Remak's observations above quoted, as well as by a remarkable case of Billroth, of a tumour of this nature in the testicle (Virch. Archiv, viii.). The best illustration of this form of cancer is to be found in Cruveilhier's Path. Anat., Liv. x.

through which process large masses may sometimes be thrown off, leaving irregularities in the substance of the cancer. The same condition also occurs, but to a less extent, in scirrhus. In colloid, on the other hand, it is seldom observed, and the ulcerative process, if so it can be called in this variety, consists of the rupture of the larger spaces, which thus give rise to a series of pits or depressions on the surface. The more rapid and extensive necrotic processes are sometimes a source of dangerous hæmorrhage.

The ulcers thus resulting are almost invariably distinguishable by their thickened, ragged edge, which is infiltrated and swollen by the morbid growth, and around which warty or polypoid excrescences are often formed, and also by the presence of cancer structures in their floor. Occasionally such large masses of the growth are thrown off as to have led to the idea that the morbid structure might possibly be eliminated in this manner: but evidence of a cure thus occurring is very defective, and, although cicatricial formation is sometimes found proceeding in one part, it is usually found that the cancer structures are extending in another. In some cases this sloughing action appears, however, to have restored the patency of the pyloric and cardiac orifices, after these had been previously obstructed by the growth.

The extension of the cancer through the peritoneal coat is attended with various consequences. Adhesions to neighbouring organs, and implications of the omentum in the cancerous growth, which are most common in cases of colloid, have been already alluded to.

General peritonitis[1] has sometimes been observed without rupture of the stomach. Partial peritonitis taking place in the same manner is, however, more common.

Perforation, leading to a free opening between the interior of the stomach and the cavity of the peritoneum, is less frequent in cancer than in ulcer of the stomach. The data as to the absolute frequency of this event in the former disease are, however, not sufficiently certain to allow of an absolute comparison.[2]

Adhesions to adjacent organs may, however, lead to the invasion of these by the cancer, as is observed in the case of the liver, pancreas, spleen, and the lumbar vertebræ, or to fistulous communications formed between the stomach and other parts. Gastro-colic fistula has been already stated to be much more frequent in cancer than in ulcer, while the converse proposition holds true with regard to gastro-cutaneous fistula.[3] Perforation of other portions of the intestines has also been noticed, as into the ileum;[4] and in other cases the growth extends through the diaphragm into the lungs.

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[1] Dittrich, loc. cit.

[2] Dr. Brinton has estimated the frequency of perforation in cancer as occurring in rather more than 4 per cent. In four of his cases the contents of the stomach were effused into a limited sac bounded by the peritoneum.

[3] See note 4, p. 924.

[4] Brinton, loc. cit. A remarkable case of this nature, which has its parallel among the secondary consequences of simple ulcer, is recorded by Dittrich, Prager Viertel-

The mucous membrane of the stomach in parts not invaded by cancer presents little that is characteristic. Evidences, however, of inflammatory action, sometimes existing in an acute form, are occasionally met with, and still more frequently there are seen signs of chronic forms of this process in thickenings and ash-grey pigmentation, with fatty degeneration of the glandular structures. In other cases, again, no deviations from the normal appearance and structure can be found.

The associated pathology of cancer of the stomach may be conveniently considered under the heads of the relation of the growth to similar structures occurring in other parts, and of accidental complications and secondary lesions not associated with the presence of cancerous formation.

The disease in the stomach, unless when propagated by continuity from other parts, is almost invariably primary.[1] Secondary affections[2] more commonly occur in the viscera of the abdomen than in more distant organs.[3] The extension of the growth to other adjacent viscera by adhesions has been already described.[4]

Obstructions of the vena cava and thoracic duct are among the rarer events.[5]

The non-cancerous secondary lesions may be also briefly dismissed, as they offer but few peculiarities in connexion with the special organ in question. The occurrence of peritonitis, independently of rupture of the stomach, has been already alluded to. It appears, from the observations of Dittrich, to be sometimes of a septic character, and to be occasionally associated with pleurisy and pericarditis, due probably to a similar mode of origin. Retrograde tubercle was found in the lungs in most of Dittrich's cases. This author only

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Jahresch. xix. 112, where a fistulous opening was established between the stomach and duodenum after the pyloric opening had been obstructed by the cancerous growth.

[1] Walshe, loc. cit. 279. It may occur among the phenomena of simultaneous multiple developments of the growth, and under these circumstances has been found to co-exist with similar disease in the ovaries and uterus. An almost unique case has been recorded by Cohnheim (Virch. Archiv, xxxviii. p. 142), when cancer was found in the stomach secondarily to a similar affection of the mamma. The liver and the axillary and cervical lymphatics were also implicated.

[2] These are found in about half of the cases of cancer of the stomach. The liver suffers in 25·6 per cent. of all cases (Brinton, Dittrich). In 160 cases given by Dittrich, the liver was affected 43 times; the peritoneum 22; the lungs 9; the rectum 2; and the ovary once. Brinton says that the lungs were affected in 8½ per cent., and the gastro-lymphatic glands in 25½ per cent., of 251 cases. Dittrich says of the latter, that it is only those in immediate proximity to the stomach which ordinarily suffer. Henoch, loc. cit. ii. 162, says that cancerous glands above the clavicle may sometimes aid in the diagnosis. Dr. Handfield Jones, loc. cit. 169, has given a case where the glands behind the stomach thus secondarily implicated contained columnar epithelium.

[3] Dr. Walshe says that he has never known the lungs to suffer in this manner without implication of the liver.

[4] In some cases the disease appears to spread by dissemination or contact, without adhesion, as in a case of Sir R. Carswell's, cited by Dr. Walshe, loc. cit. p. 282, where a cancerous tumour existed in the anterior abdominal wall, over, but otherwise unconnected with, a similar formation in the pylorus; and an illustration of a similar and very extensive process of dissemination over the peritoneal surface is given by Virchow, Krankhaften Geschwülste, i. 54.

[5] Dittrich, loc. cit.

mentions five instances of catarrhal pneumonia as a complication; but it is probable, from the statements of numerous other writers (though precise data are wanting), that a low inflammation of this type is exceedingly common, and frequently proves the immediate cause of the fatal issue. Among the other secondary affections may be mentioned coagulation of the blood in the veins of the extremities, with the phenomena of phlegmasia dolens;[1] or in the sinuses of the dura mater. Spontaneous coagulation in the arteries[2] is a less frequent occurrence. Its consequences have been already alluded to.

Ulcerations of a non-cancerous nature in the rectum and colon were noted twenty-five times by Dittrich, and endocarditis was observed in five instances.

The blood suffers markedly in its composition, especially when there is much vomiting, and still more so when there is hæmorrhage. The anæmia and waxy pallor of the complexion are largely explicable by these events; but that the interference with digestion and assimilation may take place at an earlier stage is shown by cachexia occurring, in a certain number of cases, independently of these events. Analyses of the blood have chiefly been directed to the former class, and show, as might be expected, a diminution in the number of the blood-corpuscles, and also of the total amount of solids in the serum, while the proportions of fibrine have varied, being sometimes in excess of, and at others below, the normal standard.[3]

DIAGNOSIS.—Cases of cancer of the stomach may be, as has been before stated, divided into two classes—one when the disorder runs an almost latent course, and is revealed only by failure of health and strength, by obscure dyspeptic symptoms, and by anorexia: another when it gives rise to either pain, vomiting, and hæmorrhage, singly or conjointly, and in which, at some period, a tumour can be recognised.

The former of these classes is with great difficulty distinguished from cases of atonic dyspepsia, and from some of chronic catarrh. The latter may be confounded with neuralgic conditions of the stomach, with colic, or with gastric ulcer.

It may be stated that the discovery of tumour distinctly situated in the stomach affords the only *positive* ground for the diagnosis of cancer from some other diseases of the organ. This, however, is not early apparent, and it may at times disappear, and the probability of the cancerous nature of the disease must then rest upon other data, which relatively are only of comparative value.

1. The diagnosis of the first class has been already alluded to (see Atonic Dyspepsia). The etiological conditions under which the symptoms originate are of extreme importance in estimating the nature of the disorder. If such a condition should appear without

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[1] Nine times in 160 cases: Dittrich.

[2] Twice in 160 cases: Dittrich.

[3] Andral et Gavarret, Rech. sur la Composition du Sang, p. 238. I have in one case observed a distinct increase of the white corpuscles of the blood, but there was in this instance a considerable non-cancerous enlargement of the spleen.

manifest cause after the age of thirty-five or forty, it is sufficient to excite suspicions of its real nature, especially if loss of appetite form a prominent feature. The addition of vomiting to the above, even if unaccompanied by pain, when alcoholic excesses, albuminuria, or cirrhosis of the liver can be excluded, would add additional gravity to the suspicions entertained, since both atonic dyspepsia and simple chronic catarrh are rarely associated with this symptom, except (in the case of the latter) under some of these attendant circumstances.

2. In cases where pain forms a prominent feature, unaccompanied by vomiting, by marked disturbance of the digestion, or by hæmorrhage, and when no tumour can be discovered, the diagnosis from *purely neuralgic conditions* must also mainly depend on etiological circumstances, since the characters of the pain in both these classes of disease bear a great similarity to one another, being in both usually unaggravated by food, and not associated with tenderness on pressure. Sex is one of the most important of these; sex and age collectively may, in cases where the combination occurs in a male past middle life, be of great importance, for neuralgic pains of the stomach are rare under these circumstances, being most common in females at the earlier periods of life. In the female sex these conditions afford less assistance. When the climacteric period is past, and the hysterical diathesis can be excluded, there may, under such circumstances, be some grounds of suspicion, but certainty can rarely be attained without some of the concomitant phenomena before alluded to.[1]

3. A third class, of gradual and progressive emaciation, without distinct causes of marasmus, occurring at middle life, though unattended by any distinct stomach symptoms, should also engage attention. The manner in which cancer of the stomach can thus profoundly impair the nutritive processes, though completely unexplained,[2] has nevertheless been distinctly recognised.

4. In the absence of a tumour, the diagnosis of cancer from *ulcer* of the stomach is sometimes a question of difficulty. In the majority of such cases the diagnosis is rather a balance of probabilities than a question of absolute certainty, which, however, may be attained with a greater or less accuracy in a certain proportion of instances.

Common to both diseases are the symptoms of pain, vomiting, hæmorrhage, and cachexia, and disturbances of digestion and of the secretions of the stomach, but they appear in a different manner, and at different periods of the two disorders.

The circumstances influencing the diagnosis may be briefly contrasted as follows :—

Etiology.—Ulcer is more common at the earlier periods of life, and especially so in the female sex: cancer is almost limited to the later periods of life, and its appearance is comparatively uninfluenced by sexual conditions. The pain frequently presents in both disorders a paroxysmal character, but in cases of cancer it is usually more continuous, is less influenced by food, and is less frequently relieved by vomiting. The specific character of the pain affords no positive data for distinction in either disease.

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[1] Constant and repeated experience has shown that such cases are not merely hypothetical.

[2] The condition is evidently something more than the so-called cancerous cachexia, which is rarely, if ever, observed in cancer of external parts until ulceration has set in, or unless pain has been severe.

Tenderness on pressure is most common in earlier stages of ulcer. It is rarer in a localized form in cancer until a tumour has appeared, and even under these circumstances it is less distinct. The position of either disease in the stomach necessarily influences the diagnosis in this respect.

Hæmorrhage is, on the whole, rather more frequent in cancer than ulcer, but the amount of blood effused varies considerably in the two disorders. Large hæmorrhages are much the most frequent in ulcer,[1] while smaller hæmorrhages of coffee-ground or sooty matter mixed with mucus are most common in cancer. Hæmorrhages in the earlier stages are much rarer in cases of cancer than of ulcer. Hence, in any given case, the entire absence of hæmorrhage is somewhat in favour of ulcer; the presence of small hæmorrhage alone is in favour of cancer; and the presence of large hæmorrhage alone, or occurring early in the disease, is in favour of ulcer.

Cachexia and failure of strength is a much earlier symptom in cancer than in ulcer. In the latter it is usually proportioned to the pain, hæmorrhage, or vomiting; in the former it occurs more or less independently of these conditions. The character of the cachexia appears *per se* to offer but few positive criteria.

The appetite often presents a marked difference. It is often absent or capricious in cancer; in ulcer, on the other hand, it is frequently excessive.

Pyrosis and acidity, and the vomiting of glairy mucus, and conditions of indigestion, flatulency, and constipation, are common to both diseases, and offer but few distinctions, but they are somewhat more distinct in the early stages of cancer than of ulcer.

The duration and course of the two disorders are, however, markedly different. That of ulcer may be almost indefinite. It is interrupted by long intermissions, and seldom presents a distinct uniform deterioration of the health and strength. The progress of cancer, on the other hand, from the period that the more distinct symptoms have declared themselves, is rapid, tending to a fatal issue within one or two years. The intermissions, also, when they occur, are of shorter duration, and are seldom complete. Exceptional instances at times occur, but they are insufficient to invalidate the general rule observed.

Perforation of the colon is, as before stated, in favour of the cancerous nature of the disease.

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[1] They occur in one-third of all cases of ulcer, and in only about 1 per cent. of cases of cancer (Brinton).

Dilatation of the stomach and obstruction of the orifices, together with the vomiting of sarcinæ, when occurring to any marked extent are more common in cancer than in ulcer; but this, like other points of comparison, is only true as a question of degree.

5. After the appearance of a tumour, the diagnosis becomes more definite; the fallacies, however, of its position and occasional disappearance, which have been before alluded to, require to be recollected. Ulcers of the anterior surface and of the pyloric extremity are occasionally attended with a degree of thickening, which may give rise to a fallacious sensation of a tumour; but in these cases the induration is not so distinctly circumscribed, nor is the resonance on percussion so absolutely dull, as in cases of gastric cancer. The other peculiarities arising from the infiltration of the coats in the latter disease have been before described.

Other fallacies, however, exist in cases of cancerous tumours of the lymphatics, of the small omentum, of the liver, of the head of the pancreas, and of the peritoneum and great omentum, which may at times perforate the stomach from without. When this event does not occur, the diagnosis must depend on the absence of the more prominent symptoms referable to the stomach: if the stomach is implicated, the diagnosis must become practically almost impossible without further details in the history of the case, which lie beyond the scope of this section.

It is only necessary to allude briefly to the possible fallacy of spasm of the first division of the rectus muscle simulating a tumour. This can be distinguished by its quadrangular shape, its superficial character, and by the resonance on percussion; which, however, is sometimes masked by the tension of the muscle.

6. The diagnosis of the pain of cancer of the stomach from attacks of *colic* is often, in its earlier stages, one of great difficulty. The distinction mainly depends upon the same data as serve to distinguish attacks of the latter affection from cases of ulcer (see p. 942).

THE TREATMENT of cancer of the stomach can, unfortunately, be only palliative. In their main features the indications are almost identical, both in diet and medicinal treatment, with those laid down for cases of ulcer; but as food has less influence on the pain, a rigid diet is not to be persisted in so strenuously, when it fails to relieve. Still, however, small meals of easily assimilable food, presented for the most part in a fluid form, prove in many cases very advantageous: a moderate use of alcoholic stimulants is, however, in most cases beneficial. For the relief of pain, opium is the most effectual remedy, and in some cases, when the growth has formed adhesions with the anterior wall, I have found hypodermic injections of morphia of great benefit. Fomentations, leeches, and even blisters to the epigastrium, are also of service. Vomiting is most effectually controlled by ice; but effervescents, hydrocyanic acid, and creosote may at other times be employed with advantage. Offensive eructations associated with sloughing of the tumour are often mitigated by the use of charcoal.

Dr. Walshe states that he has found a combination of trisnitrate of bismuth with extracts of hop, conium, and stramonium in pill, more useful than any other medicine. He also recommends the oil of cajeput for the relief of flatulence. Constipation may be remedied by enemata, but purgatives by the mouth should be as far as practicable avoided.

The treatment of hæmorrhage must be conducted on the principles already laid down. The vomiting of sarcinæ may at times be controlled by the administration of the alkaline hyposulphites.

VIII.—HÆMORRHAGE FROM THE STOMACH.

SYNONYM.—Hæmatemesis.

Hæmorrhage from the stomach is usually only a symptom or a result of some other disease of the mucous membrane, or of a condition of congestion induced by disorders in the portal circulation by causes extrinsic to this viscus. It will only, therefore, be necessary to give a brief retrospect of these, many of which have been already alluded to.

Recent researches have indeed proved that the older theories of hæmorrhage by exhalation are not altogether so incorrect as they have been lately believed to be, and that in cases of congestion from mechanical causes an escape of the blood-corpuscles from the capillaries is possible. In most cases, however, of this nature as affecting the stomach, the rupture of the minuter vessels of the mucous membrane must be regarded as the most probable condition; since the large hæmorrhages arising from cirrhosis of the liver or diseases of the portal system can scarcely be explained by a simple transudation. The pallor of the mucous membrane found in some cases after death affords no argument against this explanation, nor does the fact that no source for the effused blood is discoverable. The causes of this post-mortem pallor, when congestion has pre-existed, have been already considered.

In some cases the rupture of the capillaries is associated with hæmorrhagic erosions, the appearance of which has been already described.[1]

Hæmorrhages from the stomach may, therefore, be divided into two main classes, viz.: (*a*) those in which it proceeds from larger vessels, and (*b*) those in which it is derived from capillary sources.[2]

Perforation of large vessels may arise from mechanical causes,[3] or from the rupture of vessels through atheromatous changes;[4] but the most frequent, if not the exclusive, sources of hæmorrhage of this kind arise in the progress of the chronic ulcer and of cancer, to which no further allusion appears necessary.

Hæmorrhage from congestion may arise when this is simple and passive. It is a very common complication of obstructions of the portal circulation, especially by cirrhosis or acute atrophy of the liver, or by thrombosis of the portal vein,[5] which are among the conditions which give rise to the largest hæmorrhages from this cause, and some of which have been found attended by a varicose condition of the veins of the stomach. Diseases of the heart and lungs, and asphyxiating conditions, such as epilepsy[1] and strangulation, are also causes of hæmorrhage less extensive in amount than those before cited. It is probably to obstructions in the portal, cardiac, or pulmonary circulation that the hæmatemesis and melæna of newly-born children are due, though in some cases the existence of a deficiency in the nutrition and elasticity of the capillaries is evidenced by the co-existence of a hæmorrhagic diathesis, either hereditary or not.[2]

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[1] See Dr. Bastian, Trans. Path. Soc. vol. xix. 1868.

[2] Another and rarer cause has been noticed in the bursting of an aneurism of the cœliac axis into the stomach (Bamberger, loc. cit. 246).

[3] As in the case of a cork swallowed; Velpeau, Mém. Acad. de Méd. (Budd, 277).

[4] Copland, Med. Dict. ii. 93.

[5] For illustrations of all these forms, see Frerichs, Dis. of the Liver. An interesting case of hæmorrhage, from portal thrombosis, is recorded by Dr. Andrew Clark, Path. Soc. Trans. 1867, p. 61.

In other cases, also, there is probably, in addition to congestion, some alteration in the coats of the capillaries, as in diseases of the spleen, where, however, the mechanism of its origin is not fully explained.[3] In the same manner are probably produced the hæmorrhages of yellow fever, and of other malignant intermittents, as also those which occur in relapsing fever,[4] typhus fever,[5] cholera,[6] purpura, scurvy, and hæmorrhagic variola.

In other cases, though probably referable to the same source, its mode of origin is less explicable; as when it follows severe surgical operations,[7] or blows upon the back or epigastrium,[8] or even a remarkable case reported by Empis where the invasion of tubercular meningitis was associated with uncontrollable vomiting with hæmatemesis.[1] In the same class belong also hæmatemeses, occurring vicariously, of the menstrual period, which however, when independent of ulceration, seem to be less frequent than was at one time supposed.[2] In other cases, also, it may arise through the mechanism of embolism and thrombosis. (See Ulcer.)

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[1] Budd, loc. cit. 51; Yellowly, Med.-Chir. Trans. iv.

[2] A very complete account of this affection is to be found in Barthez and Rilliet, Malad. des Enfants, ii. 309.

[3] When the hæmorrhage occurs in connexion with disease of the spleen, it has usually been associated with enlargement of that organ, as the result of ague; but a case of hæmatemesis is given by Dr. Watson, Edin. Med. Journ. June 1858, where in addition to splenic enlargement there was an abnormal distribution of the splenic vein, together with an obstruction from phlebolithes in its exterior. In a large number of these cases the liver is simultaneously diseased; but that this is not always present is shown by a case by Dr. Budd, loc. cit. p. 70. The cause in some cases is probably a complex one, for other hæmorrhages, as into the skin (Piorry), or from the nose, which was noticed by Hippocrates ('Επιδημίων, ii. § 165, Kuhn's Ed. iii. 450), are very common. These have also been witnessed by recent observers, as by Bamberger, loc. cit. 654; and though it is difficult to believe that any alteration in the composition of the blood, except when attended with complete breaking down of the red corpuscles (which has not been shown to take place), can permit of its escape from the capillary walls, yet it is very probable that the nutrition of the vascular system may, in these cases, be so profoundly affected as to cause their easy rupture under slight pressure. In some cases, as believed by Siebert (Henoch, loc. cit. i. 54), the contractility of the splenic tissue may contribute to the result, as a rapid diminution of the size of the spleen has been observed after copious hæmorrhage of this nature.

[4] Murchison, Continued Fevers, pp. 336–7.

[5] Buhl, loc. cit. 68.

[6] Buhl, Report of the Munich Commission on the Cholera Epidemic of 1854, p. 500.

[7] A case of this nature was communicated to me by my friend and colleague, Mr. Berkeley Hill, when after an operation severe hæmatemesis occurred, for which after death no cause could be discovered. Sir W. Jenner informs me that he has seen similar instances. In relation to it may be recalled the observations of Rokitanski upon hæmorrhage from the bowels after severe burns (Path. Anat. iii. 200).

[8] Dr. King Chambers, Indigestions, p. 190. Bamberger, loc. cit. 245. Another is quoted by Henoch, i. 307. It is possible that, in some of these cases, laceration of the mucous membrane, without rupture of the other coats, may be the cause of such hæmorrhage. An instance of this nature is recorded by Dr. Wilks, Path. Anat. 1857, p. 275. Laceration of the *mucous membrane* of the stomach from the dragging of an omental hernia is described by Rokitanski, Path. Anat. iii. 162.

Diagnosis.—It must be recollected that blood vomited does not always proceed from the stomach, but may have been swallowed after having been effused from the nose, mouth and throat, œsophagus or lungs. The characters vary with the amount and rapidity of the hæmorrhage, and with the length of time which it has remained in the stomach.

In larger hæmorrhages, when the blood is rapidly poured into the stomach and quickly ejected, it may be coagulated, and may retain its normal colour, or may be only slightly blackened by the gastric juice.

Blood more slowly effused is acted on by the gastric juice, which prevents its coagulation, and frequently dissolves the envelopes of the red corpuscles, so that under the microscope only flakes or granules of pigment may be discoverable. In other cases the corpuscles are shrivelled, and irregular in form and size. The colour of the blood is then changed to a rusty chocolate brown or coffee-ground tint, and it is sometimes of a tarry consistence. Sometimes the altered corpuscles and pigment granules sink to the bottom of the vessel, leaving a clear supernatant fluid.

The presence of the blood can, however, be generally recognised: for bile seldom loses its more characteristic tint, nor does it assume the coffee-ground appearance just described. Food stained by ferruginous medicines will sometimes present a similar appearance; but microscopic and, if need be, chemical examination will then suffice for its distinction.

Blood, however, effused from the stomach is not invariably vomited, but may pass into the intestines and be voided by stool (melæna).

Difficulties may, under such circumstances, arise in deciding from what portion of the canal the blood has proceeded. Independently of distinct evidence of local disease, this may not always be possible, since blood proceeding from the upper part of the intestines has usually the same characters when voided by stool as that derived from the stomach. Blood from the duodenum may also be regurgitated into the stomach, and be evacuated by vomiting.

Cruveilhier was of opinion that blood effused from the stomach retained its rusty tint throughout, while that originating from the intestines was of a more inky character; but this distinction is not always applicable. The chief means of distinction depends on the more or less intimate intermixture of the blood with the fæcal matter, which diminishes in proportion as its source approaches the lower portion of the canal.[1] The existence of disease in the latter (ulcerations from tubercle, typhoid, dysentery, cancer, or the presence of hæmorrhoids) also facilitates the diagnosis. In some cases, as in disease of the liver, we must often remain in doubt, since this cause may give rise to hæmorrhage from any part of the tract.

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[1] De la Granulie, p. 154.

[2] A very remarkable case of this kind is, however, given by Sir T. Watson, Prin. Pract. Phys. ii. 425, where vicarious menstruation recurred regularly, ceased with pregnancy and lactation, and returned after weaning. He quotes also from Mr. North two other fatal cases. Dr. Murchison has cited another of older date (1712), Med.-Chir. Trans. xli. p. 46; and Henoch, loc. cit. i. 57, has observed the same phenomenon coincidently with an acute swelling of the spleen, which disappeared after hæmorrhage from the stomach and intestines.

The characters of altered bile in the stools are to be distinguished by dilution with water. Blood under these circumstances gives a redder tint.[2] Bile pigment can sometimes be distinguished by nitric acid; but bile seldom produces either the tarry or rusty appearance derived from altered blood.

The blackening of the fæces from ferruginous and other metallic preparations can usually be distinguished by the tint, and also by the history of the case.

The distinction between hæmorrhage from the lungs and that from the stomach is not always easy. Difficulty may arise either when blood is vomited immediately after its effusion in the stomach, so as to escape changes from the gastric juice, or when that proceeding from the lungs has been swallowed, and subsequently vomited in an altered condition.

In some cases, also, of mitral disease, blood proceeding from the lungs has been known to present a "bistre" or sooty tint.[3] In exceptional instances there may also be a double fallacy in the expulsive act attending each condition, for hæmatemesis may sometimes give rise to cough, and hæmoptysis may occasionally excite vomiting. The criteria ordinarily laid down are, however, usually sufficient when a patient is under observation, though it is sometimes more difficult to draw positive conclusions from the history of past attacks.

Blood proceeding from the lungs is generally frothy, aërated, non-coagulated, florid in colour, and alkaline in reaction.

That derived from the stomach is either blackened and rusty, or it may be coagulated and altered externally. If it has been at all delayed in the viscus, and unless it is in excessive quantities, it is acid.

Hæmorrhage from the lungs is preceded or attended by a sense of weight and oppresion in the thorax, together with a sense of dyspnœa and tickling cough, or with a sense of bubbling in the chest. Tinged sputa often precede and almost constantly follow the larger flow, and these are brought up by coughing, are frothy and aërated, and mixed with mucus or pus. It may be the first symptom in a certain proportion of cases,[4] but physical examination of the lungs and heart will, almost without exception, disclose evidence of disease in these parts.

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[1] Bamberger, loc. cit. 252.

[2] Ibid. 251.

[3] Walshe, Diseases of the Lungs, 416.

[4] See Ibid.

Hæmorrhage from the stomach is seldom, if ever, the *first* symptom of disease of this organ. It has usually been preceded by dyspeptic symptoms, or in the vast majority of instances by pain, nausea, or vomiting. The attack is preceded by nausea, and the expulsive act is almost invariably distinctly one of vomiting; it is not followed by cough or bloody sputa. Examination of the abdomen will usually reveal gastric tenderness, or the signs of disease of the liver or spleen.

The diagnosis of the disorders of the stomach in which the hæmorrhage originates must be determined by their distinguishing features, which have been previously considered.

THE SYMPTOMS of hæmorrhage from the stomach have been already described (see Ulcer of the Stomach).

PROGNOSIS.—Severe hæmorrhages from the stomach are occasionally directly fatal; the author's conviction is, that this is more frequently the case when they arise from cirrhosis of the liver than when originating from ulcer or cancer of the stomach. In the latter disorders, however, they contribute largely to a finally fatal result by the exhaustion and anæmia which they induce.

THE TREATMENT consists of rest and the administration of hæmostatics.

In some cases, when the hæmorrhage proceeds from congestion through obstructed venous return, particularly when the cause resides in the liver, it is benefited by purgatives;[1] but these are strongly contra-indicated in cases of ulcer and cancer. With the above exceptions, cold and astringents must be resorted to. Ice, in small pieces, may be sucked constantly. Turpentine, acetate of lead, tannic acid, the perchloride of iron, or alum, or the infusion of matico, may also be tried. My own experience leads me strongly to prefer the acetate of lead in the majority of cases.

When the hæmorrhage is severe, the head must be kept low. Brandy may be administered by the rectum, or ether inhaled. If temporarily arrested, abstinence from food should be practised as completely as possible for some time, and nutriment should only be given in a fluid form, in very small quantities, and cold, so as to avoid both the afflux of blood during the digestive act, and also all movement of the stomach.

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[1] Sir T. Watson, loc. cit. ii. 435.

IX.—HYPERTROPHY OF THE WALLS OF THE STOMACH.

SYNONYMS.—Cirrhosis; Plastic Linitis (Brinton); Fibroid Induration (Handfield Jones); Sclerosis (Snellen).

Thickening of the coats of the stomach appears to be a rare disease, and one that is at present but little associated with any definite group of clinical symptoms. It is also one regarding which much confusion has existed, and on the nature of which some doubt still remains in the writings of pathologists.

Andral, who furnished the first systematic description of it,[1] confounded it with scirrhous induration; or, rather, he described that form of cancer in the stomach as consisting only of a thickening of the coats of the organ, induced by chronic inflammation—an opinion which has been further supported by Bruch.[2] Hypertrophy of the muscular coats is a common result of pyloric obstruction.[3] Cases of general thickening to any extreme degree of the coats of the stomach, independently of such causes, though occasionally met with, are nevertheless of extreme rarity.

Thickening of the mucous membrane from chronic catarrh has been already described; but this condition does not usually invade the submucous tissue, nor is the muscular coat generally affected.

In the cases, however, described under this head, a general thickening of all the coats ensues, which, however, especially affects the muscular and submucous layers. In some cases this is found more particularly in the pyloric region, under which circumstances that orifice is usually considerably narrowed. In other instances it implicates to a greater or less degree the whole of the organ.

In some cases the thickening has apparently resulted from abnormal growth of fibrous tissue, which has indurated the coats, and caused wasting of the muscular substance, but in which no evidence of a cancerous nature has been found by microscopic examination.[4] Rokitanski considers that this change may sometimes be the final result of suppurative inflammation in the submucous tissues.

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[1] Prec. Path. Anat.

[2] Zeitsch. Rat. Med. 1849.

[3] Louis, Rech. Anat. Path. p. 121 et seq.

[4] See a case of this kind reported by Dr. Hare and examined by Dr. Lionel Beale, Path. Soc. Trans. iv. 129. Another case reported by Dr. Quain, but in more doubtful terms, is in the same volume. Another case is reported by Dr. Handfield Jones, Stomach, p. 121. A preparation of great hypertrophy of the muscular and subserous coats, with no history attached, is in the museum of University College. Four cases are also given by Dr. Hughes Bennett, Cancerous and Cancroid Growths; and another by Dr. Wilks, Path. Soc. Trans. xiii. 83. In Dr. Wilks' case a similar change was found in the intestines. Two cases of the affection limited to the pylorus are recorded by Dr. Habershon, Obs. Alim. Canal, 1857, p. 99. Several cases are also recorded by Brand, Ueber Stenosen des Pylorus, Diss. Inaug.; Erlangen, 1851. Also a case by Snellen, Canstatt, Jahresb. 1856, iii. 302, where the disease followed an injury to the epigastric region and affected the whole stomach.

The coats of the stomach may, under these circumstances, attain a thickness of an inch or an inch and a half. The mucous membrane is thrown into folds, and in some cases has been found thinner than natural (Dr. H. Jones). The cavity of the stomach, in a case recorded by Dr. Hare, was much contracted, so as only to contain about four ounces of fluid.

The symptoms in the recorded cases have varied, and have been complicated with the presence of ascites (Dr. Wilks), and with recent peritoneal inflammation (Dr. Hare and Dr. Wilks); or, when the disease has been limited to the pylorus, with the signs of obstruction. Vomiting, which has sometimes been attended with the ejection of matter of coffee-ground appearance, has been noticed in some instances; and pain, but not of a severe kind, has usually been present. Gradual emaciation has also been observed. The cases have generally been chronic; in some instances proceeding to a fatal termination in two or three years. In some cases the stomach has formed a distinct tumour, perceptible through the parietes, but which has, however, been resonant on percussion (Louis and Dr. Hare).

The causes of this condition, independently of the observation of Rokitanski, are very obscure, and, from its extreme rarity, the disorder must be considered at present to be one which presents features rather of pathological than of clinical interest.

The main features by which it can be pathologically distinguished from scirrhus of the stomach have been already alluded to. (See note 1, p. 960.)

X.—STRICTURE AND OBSTRUCTION OF THE CARDIAC ORIFICE OF THE STOMACH.

SYNONYM.—Stenosis of the Cardia.

This affection is comparatively rare, except when caused by cancerous growths occluding or contracting the orifice. Simple spasm appears occasionally to cause temporary obstruction of this nature. It may, however, result from the cicatrices of simple ulcers, or of such as have been caused by swallowing corrosive poisons. Foreign bodies impacted at the cardiac orifice may, it is said, act as a cause.[1] Obstructions may also result from aneurismal or other tumours pressing on the œsophagus at any part of its course. The symptoms of such pressure are almost identical with those of occlusion from disease of the canal.

The primary effect of these obstructions is the regurgitation of food into the mouth. The secondary consequences are those of starvation, proceeding more or less rapidly according to the degree of obstruction present.

The regurgitation into the mouth of the food delayed at the cardia takes place by an act resembling vomiting. The characters of the food thus returned depend on the degree of obstruction, or on the amount of dilatation which the œsophagus has undergone.

When the obstruction is incomplete, fluids may pass when solids are returned. A degree of spasm, or sometimes of paralysis, is often combined with the mechanical obstacle, since the facility of swallowing varies at different periods; and a narrow tube can sometimes be passed through the obstruction, by which patients may be fed, when all the food appears to be regurgitated. When dilatation of the œsophagus has not ensued, the food is very quickly returned, almost unaltered, except by mastication. When, however, the increased calibre and diminished muscular powers of the œsophagus above the seat of the obstruction admit of its retention, it becomes changed by maceration, or it may undergo fermentation or putrescent changes, which give rise to offensive eructations, and the matters ejected are often covered or mixed with a quantity of tenacious mucus.

Pain is generally felt, especially during deglutition: it is commonly referred to the ensiform cartilage or mid-dorsal region. The pain is seldom very severe, except when ulceration is present. In the intervals of deglutition there is often a dull sense of uneasiness. In many cases the patients are distinctly conscious of the point where the passage of the food is arrested.

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[1] Bamberger, loc. cit. The author desires to express his obligation to this writer for much of the systematised information contained in this and the ensuing section.

The passage of a bougie will almost certainly indicate the point of obstruction; but this should be cautiously practised, owing to the danger of laceration of the œsophagus. The appetite is unimpaired, and patients often suffer intensely from hunger and thirst. Emaciation proceeds *pari passu* with the degree of obstruction. Dropsy of the lower extremities supervenes in some cases. Hectic is occasionally observed.

The termination is almost invariably fatal, except in cases of simple cicatricial narrowing, where dilatation may sometimes be practised. Death finally ensues by asthenia, or by pneumonia, or gangrene of the lung, or by rupture of the œsophagus.

THE PATHOLOGY of these cases depends on the discovery of the cause. In most instances of cancerous obstruction of the orifice the obstruction is due to scirrhus. Higher in the œsophagus, epithelial cancers are an equally frequent cause.

The stomach, in the later stages of the disease, is often greatly diminished in size, so as not to exceed that of the intestines. No special alteration of its mucous membrane has been recorded.

THE DIAGNOSIS of the obstruction is usually easy. It is determined by the character of the food regurgitated, by the obstruction felt in swallowing, and by auscultation during the act of swallowing. The passage of the bougie serves to distinguish between the effects of organic stricture and of simple spasm, and also to determine the site of the obstruction. Percussion will occasionally ascertain the existence of dilatation of the œsophagus. The diagnosis of the cause of the obstruction must in a great measure depend upon the history of the case. Spasmodic obstruction is intermittent, and is almost invariably associated with the hysterical diathesis. Simple cicatricial contractions are very rare, except when corrosive fluids have been swallowed. The regurgitation of blood or pus,[1] or in rarer cases the discrimination of cancer cells in the matters vomited, would indicate, in all probability, the malignant nature of the obstruction.

THE TREATMENT consists in administering food of nutritious properties in a liquid form. The fact that, in some cases, a tube can be passed, will suggest this mode of giving nourishment. Nutritive enemata may also be administered. Thirst may in some persons be allayed by baths. Opium is also of value in diminishing the sufferings of the patient: it may be given in small quantities, by the mouth, by enemata, and by the hypodermic method. Dilatation may in some cases be successful, when the stricture is clearly traceable to cicatricial contractions resulting from corrosive poisons. Dilatation by the bougie, bismuth, the valerianate of zinc, small doses of strychnine, bromide of ammonium, and iron, are useful in hysterical cases.

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[1] Dr. King Chambers: see *ante*, Vomiting.

The attempt to relieve the patient by gastrotomy and the establishment of a gastric fistula has not hitherto proved successful, but it would appear on many accounts deserving of a trial; though in the majority of the cases which result from cancerous growths, the fatal termination could only be delayed by such a procedure.[1]

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[1] A successful case of gastrotomy, undertaken to remove from the stomach a bar of lead which had been swallowed, is recorded by Mr. Bell, Med. Times and Gaz. March 31, 1860. Another for the removal of a knife is quoted by Mr. Gray, Holmes, Syst. Surg. ii. 338. In this article a tabular statement is given of the cases where this operation has been attempted.

XI.—STRICTURE AND OBSTRUCTION OF THE PYLORUS. DILATATION OF THE STOMACH.

Stenosis, Constriction, and Obstruction of the Pyloric Orifice are comparatively rare, independently of obstructions from cancers and other tumours of the mucous membrane, or from the cicatrices resulting from the healing of ulcers, or sometimes from the effects of corrosive poisons.[1]

Pyloric stenosis may, however, result from the induration of the submucous tissue described in a previous section, which may affect the stomach throughout, or may be limited to the pyloric ring. It appears also, occasionally, to result from hypertrophy of the muscular coats limited to this portion of the organ, a form of disease which, as stated by Dittrich, appears to occur with preponderating frequency in the earlier periods of life.[2]

Whether *spasm* can continue sufficiently long to produce any of the more serious symptoms resulting from organic disease must be regarded as very questionable.

Obstruction to the exit of food from the stomach may also arise from pressure upon the pyloric orifice or first part of the duodenum, by tumours originating externally to the organ. The most common of these are cancerous growths in the pancreas, in the lymphatics of the small omentum, and in the liver. Cancer of the gall-bladder has also been observed to produce this effect.[3]

THE SYMPTOMS are essentially those resulting from obstruction to the passage of food from the stomach, though varied by those of the other diseases in which the obstruction has its origin.

Vomiting is the most distinct and prominent of these. Its appearance is not necessarily indicative of absolute closure of the pylorus, for Bamberger has observed it to occur in cases where the orifice would still admit of the passage of the little finger.[4] Its character and time of appearance, however, greatly depend on subsequent changes in the stomach, and especially on the degree of dilatation which the organ undergoes—a condition which, as it may occur (though rarely) independently of pyloric obstruction, requires a separate description.

DILATATION OF THE STOMACH.

Dilatation is a common and almost constant result of constriction of the pyloric orifice, unless this is counterbalanced by muscular hypertrophy. It may, however, result independently of any obstruction to the pylorus, under certain circumstances, which deserve consideration. Thus :—

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[1] Dr. Markham, Path. Soc. Trans. x. 160, relates a case of obstruction of the pylorus as a secondary effect of swallowing Burnet's fluid.

[2] Brand, loc. cit. p. 15.

[3] Dr. Markham, Path. Soc. Trans. viii. 243.

[4] Bamberger, loc. cit. 253.

(*a*) Weakened conditions of the muscular coats, arising from malnutrition or impaired innervation, are a very common cause of not inconsiderable dilatation of the stomach. Such conditions arise in the course of severe general diseases, such as fevers, or from local causes in cases of peritoneal inflammation. They are also common in cases of hysteria, hypochondriasis, and in some diseases of the cerebrospinal centres, and also in some instances when the probability of one or other of the latter class of causes having existed is rather a matter of inference than of positive proof.[1]

(*b*) Paralysis of the muscular coat, limited to the pyloric portion and preventing the propulsion of food into the duodenum, has also been observed to cause secondary dilatation of the stomach.[2]

(*c*) In addition to these causes, obstruction of the duodenum, or even of the upper portion of the jejunum,[3] has been followed by the same result. Excessive eating of vegetable food has been described as a cause by Dr. Hodgkin.[4] Dislocation of the stomach and dragging of the viscus by omental hernias are mentioned as causes by Bamberger.

The symptoms of this condition are divisible into two classes: firstly, the evidence of the delay of the food for an unnatural period in the stomach; and, secondly, the physical signs resulting from the dilatation of the organ. It is only in comparatively rare cases that the dilatation, when proceeding from other causes than pyloric obstruction, is accompanied by the severer objective symptoms, and, with a few exceptions, the description of these refers almost exclusively to this class.

The immediate result of the retardation in the propulsion of the food is the production of fermentative changes; which are evidenced both by the tympanitic distension of the organ, and by the matters vomited. The nature of the changes thus undergone by the food, and the products of these, have been already considered. (See *ante*, p. 811.) The matters vomited are almost invariably frothy and acid, and in the scum on the surface torulæ and sarcinæ are commonly found.[5] (See *ante*, p. 812.) The alterations in the food vary with the duration of its delay in the stomach; sometimes the last meal is retained, while previous ones are rejected,—a difference probably due to the relatively greater fluidity of the latter. The amount vomited and the length of the intervals between the act also vary in proportion to the degree of obstruction of the pylorus, or to the extent of the dilatation, or of the paralysis of the muscular coats. The vomiting generally occurs at longer periods after eating than in most of the other diseases of the stomach, though sometimes the act is much delayed both in ulcer and in gastric catarrh. When, however, the dilatation or paralysis is extreme, days may elapse between the recurrences of this symptom, but under such circumstances enormous amounts of altered food may be ejected.

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[1] See an interesting case of this class by Dr. Humby and Mr. Miller, Path. Soc. Trans. iv. 137. The invasion of the disease was comparatively sudden. There was vomiting, and the patient died after eleven days' illness. She was seen by several physicians, among others by Sir T. Watson and Dr. Bright. The stomach reached to the pubes ; the muscular coat had in some places given way, but no cause for the distension could be discovered. A somewhat similar case is recorded by Andral, Clin. Méd. ii. 122. See also Dr. Peebles, Edin. Monthly Journal, 1840, vol. liv.

[2] As in a case by Andral, Clin. Méd. ii. 117, where there was extensive ulceration of the pyloric region without obstruction, and yet extreme dilatation of the stomach. The same condition of dilatation was observed in another case, where, in conjunction with induration of the coats of the pylorus, the muscular coat was atrophied, but the pyloric opening maintained its natural size. In this case also there was dilatation. (Ib. p. 115.) Traube, Gesammelte Beitrage, ii. 988, attributes such dilatation to destruction of the branches of the pneumogastric nerve.

[3] Andral, Clin. Méd. ii. p. 129.

[4] Lect. Serous and Mucous Memb. ii. 277.

[5] It would appear not improbable that in the cases recorded by Dr. Budd and Mr. Busk, when sarcinæ appeared without evidence of pyloric obstruction, but in consequence of

Bamberger remarks that the vomiting may cease if *complete* paralysis of the muscular coat should ensue.

Eructation and heartburn are often observed to precede the vomiting, and in some cases acidity, apparently resulting from hypersecretion, has been noticed.[1]

The appetite is variable. It is sometimes considerable, or even excessive; but in some cases this is attributable to the vomiting. Emaciation is an almost constant result: the extent to which it proceeds depends on the amount of food retained and assimilated. Constipation is, as might be expected, nearly invariably present. Dropsical swellings of the lower extremities have been seen in some cases.

The physical signs are those of extreme distension of the stomach. The tympanitic note of the organ may be heard over the extent which it occupies, though sometimes, when food is present in its interior, the inferior portions may be dull on percussion, and the dulness may change in site with the position of the patient. The distension may be so great as to push the diaphragm upwards. The tympanitic resonance may reach even to the fourth rib, displacing the heart's apex upwards and forwards. Sometimes the prominence formed by the distended stomach can be perceived by the hand in the epigastric, umbilical, or pubic regions, and the shape of the organ may sometimes be recognised by the smaller curvature being well defined; and in some cases, particularly when there is hypertrophy of the muscular coats, the movements of the organ can be distinctly felt, and may be excited by external stimulants. In some cases the obstructing tumour can be perceived at the pylorus, but this, for the reasons before stated, is not constant.

Auscultation sometimes gives a splashing sound on movement, which, Bamberger says, is one of the best signs of this form of dilatation. In other cases, during drinking, the fluid may be heard and felt by the patient to pass into the large cavity. The heart's sounds may be occasionally heard to consonate in the distended organ.

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injury, that the cause of the fermentative action may have been due to a failure of the propulsive power of the stomach due to paralytic causes.

[1] Bamberger, loc. cit.

THE PATHOLOGY of this affection has been already alluded to under its etiology. The size attained by the stomach may sometimes be very considerable; it may fill the greater part of the abdominal cavity and it may reach even to the pubes.[1] The chief point of further practical interest in these cases is the condition of the muscular coat. This may sometimes be thickened, and under such circumstances the affection is less injurious to health than when it is thinned, distended, and paralysed.

DIAGNOSIS.—That of obstruction of the pyloric orifice, independently of dilatation of the stomach, or of the discovery of a tumour, is surrounded by so many fallacies that certainty would appear to be almost unattainable. The only distinctive symptom is vomiting after food; and unless dilatation is so considerable as to allow of fermentation taking place, the act is seldom delayed to a period capable of enabling a distinction to be made between it and other diseases giving rise to this symptom; for in the female sex the distinction of such cases from hysterical vomiting would be excessively difficult, and it must be recollected that in either sex cancer may run its course without pain. The duration of the case may, however, assist in the diagnosis from cancer.

The discovery of a tumour or of induration in the pyloric region, associated with persistent vomiting, are the only data on which reliance can be placed. The existence of a tumour would raise a presumption that the disease was of a cancerous nature; but a positive diagnosis would scarcely be justified without the presence of some of the more distinctive signs of this disorder.

When dilatation is present, the combination of symptoms and physical signs above given (and which it is unnecessary to repeat) is sufficient for its recognition. Extreme meteoristic distension of the abdomen might be confounded with cases of dilatation not associated with pyloric obstruction, and where vomiting is not a prominent feature. The discrimination may then be made by the methods suggested by Bamberger, of auscultation during the swallowing of liquids, and by the passage of a sound into the cavity of the stomach, in addition to the results of percussion and the splashing sounds audible on succussion of the patient.

PROGNOSIS.—The course and termination of cases of dilatation of the stomach depend very much upon its cause. Simple dilatations, without obstructions, have, however, proved fatal. When resulting from obstruction, the course of the disease is usually prolonged, though its duration is much influenced by the conditions of the obstruction. Obstructions arising from simple thickening or the cicatrization of ulcers have usually a slower progress than is observed in cases of cancer of the pylorus.

The possibility of the re-opening of the pyloric ring by sloughing processes in the latter disease has been already alluded to. Bamberger has also seen a patient recover when the obstruction was, in all probability, due to the cicatrix of an ulcer.[1]

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[1] This has been observed in cases of dilatation independent of obstruction.

Treatment.—The treatment of cases of obstruction of the pylorus can be only of a palliative nature.

One main indication is the administration of food in small quantities at frequently repeated intervals. It is also of extreme importance that the food given should be of such a nature that it can undergo its chief metamorphoses by the process of gastric digestion.[2] Hence protein substances, beef-tea, pounded meat, or, possibly, meat already digested, as suggested by Drs. Marcet and Pavy, should form the chief articles of diet. Milk should be used more sparingly. Starchy substances, which undergo acid fermentation, and oily food, should be as far as possible avoided. Nutrient enemata of beef-tea may be used with advantage. The administration of pepsine and hydrochloric acid with the food is also desirable.

Fermentation of the food associated with the vomiting of sarcinæ, which, if permitted to continue unchecked, greatly increases the distension of the stomach, and tends to weaken its muscular coats, may be greatly controlled by the administration of the alkaline sulphites and hyposulphites recommended by Sir W. Jenner.[3]

Creosote and carbolic acid are less useful in this respect; these remedies have appeared to me to be capable of delaying, in some cases, the normal gastric digestion.

Alkalies and antacids are useful in relieving the acidity resulting from the fermentative processes. The other indications for treatment must depend on the nature and character of the obstruction. The chief of these have been already given in the chapters on Ulcer and Cancer.

The indications for the treatment of *dilatation of the stomach occurring independently of pyloric obstruction* may be summarized as consisting in the avoidance of further distension, and in restoring the tone and contractility of its muscular fibres.

Large meals should be avoided, and the food, as in the other form of dilatation, should be digestible and easily assimilable. Fermentation is also to be controlled by the means above indicated;[4] and carminatives, such as cajeput, aniseed, or camomile, may sometimes give relief.

The use of strychnia, and the taking of ice in small quantities, are the methods chiefly to be relied on for the second indication. Iron may also be used with advantage when anæmia is present. Cold douches to the abdomen or spine, and galvanism to the abdomen, would appear to be deserving of a trial.

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[1] Loc. cit. p. 236, note.

[2] Kuhne has shown that when the pylorus is ligatured, the whole of the food, if suited to the condition of gastric digestion, may disappear from the stomach (Lehrb. der Phys. Chemie, p. 52).

[3] Med. Times and Gaz., Aug. 1851.

[4] Dr. Budd narrates a case where a patient who had long suffered from the vomiting of sarcinæ obtained great relief by taking large quantities of common salt (Stomach, p. 234).

In addition to these measures, Dr. Kussmaul, of Freiburg, has recently practised with success, in cases of this nature, a method based on the analogy afforded by the beneficial effects resulting from the complete evacuation of the paralysed and distended urinary bladder, by means of the catheter. Dr. Kussmaul, however, remarks that a true paralysis of the muscular coats of the stomach does not exist in most cases of dilatation, and particularly in those resulting from pyloric obstruction, since in most of these the movements of the distended viscus can be both seen and felt through the abdominal parietes. The power of evacuating its contents is, however, lost; the passage through the pylorus, even when a considerable degree of patency is found *post mortem* to have existed at this orifice, being further impeded by the displacement, and possibly by the torsion, of the opening. Dr. Kussmaul purposes to evacuate the stomach by means of the stomach pump, whenever there is evidence of any considerable accumulation of food in its interior, and in particular when vomiting has commenced.[1] It would appear that even a frequent repetition of the act of vomiting may fail under these circumstances completely to evacuate the fermenting contents of the stomach, and that, after it has taken place, a large quantity may still be removed artificially. After the complete evacuation has been effected, Dr. Kussmaul purposes to neutralize the acid mucus, still charged with products of fermentation, by washing the stomach out with Vichy water, and other remedies can thus be applied with a similar object, such as sulphurous acid, solutions of carbolic acid, or borax. Dr. Kussmaul has found this method practically curative in some cases, when from the symptoms present it appeared probable that the dilatation of the stomach resulted from a partial stricture of the pylorus succeeding an ulcer of this part. The muscular power appeared gradually to be restored when distension was thus prevented. The indication for the employment of this measure is considered by Dr. Kussmaul to be the return of vomiting or of regurgitation; but several days frequently elapsed before the necessity for their repetition occurred, the intervals gradually becoming longer in proportion as the muscular coats regained their tonicity. Cases of cancerous stricture were also in some measure relieved by this procedure. The prevention of the distension of the stomach appears to be aided by the application of an abdominal bandage beneath its lower border. Care in diet (of which milk and meat should form the principal ingredients) is necessary to avoid further distension.

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[1] Ueber die Behandlung der Magen-Erweiterung durch eine neue Methode; Freiburg, 1869. The stomach pump employed is one invented by Dr. Wyman in America, and described by Dr. Bowditch, Amer. Journ. Med. Sciences, N.S. xxiii. 1852, p. 320.

XII.—Softening of the Stomach.

Synonyms.—Post-mortem Softening of the Stomach, Self-Digestion of the Stomach; Gastromalakia; Ramollissement de l'Estomac; Magen-Erweichung. *Var.* Ramollissement simple; R. pultacé; R. gélatiniforme.

Softening of the coats of the stomach has been already described as one of the results of recent inflammatory action. The condition produced by this process has been spoken of as being analogous to the softenings occurring under similar circumstances in other tissues, and the appearances which it presents have been defined as consisting of *swelling* and *increased opacity* of the membrane, together with a diminution of its consistence extending for a variable depth in its substance.

Other forms of softening are, however, met with, when the membrane is of a pulpy consistence, breaking under the slightest touch; but when, instead of being opaque, it is *transparent*, more or less *gelatinous* looking, and generally, although not invariably, thinner than natural.

The nature and causes of these conditions have been much disputed; and as the opinions held respecting their true character have exercised no inconsiderable influence on the pathology and diagnosis of the disorders of the stomach, it appears desirable to devote some consideration to their real significance and to their mode of origin.

Softening, thinning, and even absence, of considerable portions of the lining membrane of the stomach, had been noticed by Morgagni,[1] but the first attempt to explain these appearances was made by John Hunter,[2] who attributed such changes to the self-digestion of the stomach after death, which he regarded as resulting from the action of the gastric juice on its coats, and which he believed was prevented during life by the influence of the vital principle. He confirmed his observations on the human subject by experiments on fishes, which shortly after received a further support from Spallanzani,[3] who also observed that the digestion of food was continued after death. These observations were continued, and Hunter's views were confirmed by Adams,[4] Allan Burns,[5] Wilson Philip,[6] Gairdner,[7] and Carswell;[8] and there is a considerable uniformity in their descriptions of the appearances observed, and which they attributed to the same cause.

Stomachs in this state present, usually at the fundus, a portion where the membrane is evidently thinner than natural, or is entirely absent over an area of variable extent, while for some distance around it is softened and pulpy. The transparency of the tissue is greatly increased, allowing the white submucous coat to appear through the membrane, while the colour varies with the quantity of blood contained in the part. If this is small, the coats of the stomach are of a bluish white; if it is more considerable in amount, they are brownish or blackish. The vessels also may be seen of a blackened colour, ramifying through the affected area, and their blackened contents may often be expressed in drops from their eroded ends.

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[1] De Caus. et Sed. Morborum, Epist. xxv. 2, xliii. 22, lv. 2.

[2] Phil. Trans. 1772, republished in Obs. on certain Parts of the Animal Economy.

[3] Expériences sur la Digestion, 1783, 264.

[4] On Poisons.

[5] Edin. Med. Surg. Journ. 1800, vol. vi.

[6] On the Vital Functions, 1817.

[7] Edin. Med. Surg. Journ. 1824.

[8] Edin. Med. Surg. Journ. 1830.

Occasionally, but not commonly, very early stages of this process are observed, when the superficial layers of the mucous membrane are separating in flakes and are much softened, but not wholly dissolved. The surface of the membrane may have, under such circumstances, a somewhat whiter and more opaque appearance, corresponding to the form described by Cruveilhier as the Ramollissement pultacé.

In some cases the membrane is uniformly affected; in other instances, when the stomach is contracted, the softening affects only the prominent rugæ, leaving the intervening sulci unchanged. Occasionally, also, the coats of the stomach and of the intestines are found uniformly swollen, transparent, and jelly-like, having lost all trace of structure, and resembling albumen, or presenting appearances seen in tissues after the action of an acid.

The extent of surface over which the change in question may occur is variable, as is also its precise seat. The whole of the stomach has been found thus softened, but more usually the condition is limited to the fundus or posterior portions; while in other, but rarer, instances it is seen only in the pyloric region or on the anterior wall, while the parts above mentioned have escaped. Often the softened portion is found to be abruptly limited by a well-defined border; but in other cases it merges insensibly into the surrounding membrane.

The softening and erosion often proceeds to perforation of some parts of the stomach, intestines, or œsophagus,[1] allowing their contents to escape into the abdominal or pleural cavities, and in such cases analogous effects have been produced on contiguous viscera, especially on the spleen, liver, kidneys, or lungs; and in the last-named organs the change may be produced either subsequently to that of the diaphragm, or directly by perforation of the œsophagus, in the latter of which cases the alteration is usually found in the left side of the thorax.

The edges of the perforation of the stomach ensuing in this manner are thin, ragged, eroded, transparent, and having an appearance (to use the words of Hunter) "very much like that kind of solution which fleshy parts undergo when half digested in the living stomach, or when acted on by a caustic alkali."

Other authorities have, however, maintained that the forms of softening now described may occur during life as the result of disease.

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[1] Wilkinson King, Guy's Hosp. Rep. vii. 1842.

The chief of these have been Jaeger,[1] Camerer,[2] Cruveilhier,[3] Louis,[4] and Andral.[5] The three first named of these authors, and also Billard,[6] attributed to these supposed pathological conditions a definite set of symptoms, which are said to be met with in greatest frequency among children, but are stated to occur also occasionally among adults. Their leading characteristics are enumerated as violent fever, a semi-comatose condition, interrupted by restlessness, cries, and great irritability of temper, intense thirst, pain in the abdomen, frequent uncontrollable vomiting, diarrhœa, with grass-green stools, and early collapse, followed by death in a few hours, or in the course of a few days. It appears possible, however, from an observation of Ziemssen's, that under conditions involving the unduly long retention of the contents of the stomach in its interior in an acid condition, this effect may even be produced during life.[7]

There has been, however, a wide diversity of opinion among these authors respecting the nature of these changes, and every shade of appearance has been described as presenting a distinct pathological character, while the significance of each of these has, in almost every instance, received a different interpretation from those who have opposed the views of Hunter and Carswell.

Thus Jaeger and Camerer attributed them to disturbances in the nutrition of the stomach, induced by impaired innervation.[8] Andral, while recognising as inflammatory the form of softening usually attributed to that process, described the appearances now under consideration as resulting from a vital change, non-inflammatory in its nature, and analogous to the softenings which occur in the cornea under conditions of impaired nutrition. Louis, on the other hand, regarded as inflammatory the softening which Carswell had ascribed to the action of the gastric juice; but described also a form of "simple softening," which he attributed to cadaveric change. Cruveilhier again distinguished two forms: the "Ramollissement pultacé" and the "Ramollissement gélatiniforme;" the former being, according to him, of post-mortem origin, while the latter is caused by a vital change, "specific" in its nature, and due neither to inflammation nor to gangrene, and to which he attributed the symptoms above described as characteristic of this variety.

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[1] Ueber die Erweichung des Magens: Hufeland's Journal für prakt. Heilkunde, 1811, 1813.

[2] Versuch über die Natur der krankhaften Magen-Erweichung, 1828.

[3] Méd. Prat. éclairée par l'Anat. et Physiol. Path. 1821; Anat. Path. Liv. x.

[4] Du Ramollissement avec Amincissement de la Membrane Muqueuse Gastro-Intestinale, Rech. Anat. Path. 1826. De la Fièvre Typhoïde, 1841, 2d Edit. pp. 156, 157, 407. Phthisis, Dr. Walshe's Trans. Syd. Soc. Ed. pp. 60, 189.

[5] Préc. Anat. Path. ii. 88; Clin. Méd. ii.

[6] Maladies des Enfants, 1828.

[7] See p. 992.

[8] An opinion still maintained by Rokitanski, Path. Anat. iii. 179. This distinguished pathologist states that precisely identical appearances may be produced in the stomach by changes ensuing before and after death. He says that the softenings of the stomach met with in cases of brain disease and in the acute febrile and inflammatory diseases, are effected during life in consequence of disturbed innervation. It would appear, however, to the author, for the reasons hereafter to be stated, that the post-mortem nature of these changes is the more tenable hypothesis, though the softening thus found may be due to and may indicate perverted conditions of secretion existing during life.

Recent researches have, however, tended to confirm Hunter's opinion, and to show that the forms of softening described by Louis, Jaeger, Camerer, and Cruveilhier, and also the second form of Andral, are all to be ascribed to variations occurring in the same process of solution by the gastric juice. Cruveilhier's arguments against this view deserve, however, to be recapitulated, since they express most of the difficulties which have beset the adoption of this explanation of all the forms now under consideration; and it is chiefly to the elucidation of these that more modern observation has been directed. He stated that the theory of Hunter and Carswell was inapplicable to cases when the œsophagus, or the anterior wall of the stomach, was affected (the body having been placed after death in a recumbent posture); that the softening is sometimes found when the stomach is empty;[1] that it can only be artificially produced by a much larger amount of acid than is found *post mortem* in the stomach, and that the softening of the intestines, whose contents are normally alkaline, cannot be produced by the action of an acid.

In relation to these difficulties it may, however, be stated—

(*a*) That, as shown by the observations of Dr. Gairdner, which have been confirmed by numerous subsequent observers, both forms of softening described by Cruveilhier have been found *post mortem*, unpreceded by any of the symptoms described by him and Camerer and Jaeger as characteristic of the supposed disease.

(*b*) The appearances thus described can be artifically produced at will after death in a previously healthy stomach in any part which is most dependent.[2]

(*c*) The observations of Brücke[3] and Bernard[4] have shown that for some time after death a substance continues to be formed in the gastric follicles, having all the properties of the gastric juice; and that the acid reaction, which during life is normally only found upon the surface of the mucous membrane, may within a few hours after death extend through its whole thickness. As also the gastric juice is normally formed in greater quantities in the cardiac than in the pyloric half of the stomach, the observations of Dr. Wilkinson King[5] concerning the greater acidity of this part when the stomach has been found so softened receive a further confirmation.

(*d*) Elsaesser's[6] observations further show that fermentescible substances, whether milk or those belonging to the amylaceous series, undergo, not only during life but also after death, an acid fermentation, and become capable of producing the phenomena of softening, both simple and gelatiniform, not only in the stomach, but also in the intestines; and that when these changes have occurred in the latter organs, their contents have been found to present an acid reaction.

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[1] For a confirmation of this statement see an experiment by Dr. Blundell, quoted by Dr. Hodgkin, Morbid Anatomy of Mucous and Serous Membranes, ii. 309; where a dog which had been maintained for weeks without food, by means of the injection of blood into his veins, was nevertheless found to have softening of the stomach after death.

[2] Burns, Carswell, Camerer; also Williamson, Dublin Journal, vol. xix. 1841.

[3] Sitzbericht der Akad. der Wiss. zu Wien, xxxvii. 165.

[4] Liq. de l'Organisme, ii. 377.

[5] Guy's Hosp. Rep. 1842, vol. vii., and 2d Ser. vols. i. iv.

[6] Die Magen-Erweichung der Säuglinge, 1846.

These fermentative processes may originate in imperfect digestion, to which infants are so liable. They are, however, most easily excited through the catalytic effects of the mucus produced by catarrhal inflammation. When present, they also tend to cause the acuter forms of gastro-intestinal catarrh, the symptoms of which correspond very closely with the disorders which have been described as symptomatic of these changes in the mucous membrane, but which are really only the consequences of the inflammation thus set up. The softening, on the other hand, is on this hypothesis believed not to occur during life, but to be the result of the post-mortem action of the acids thus generated upon the coats of the stomach.[1]

(*e*) It remains to be shown why the condition is present in some cases and not in others; and perhaps among the most important of the former class must be reckoned those cases in which the membrane has been found softened, while the stomach is empty, as has been observed by Blundell, Cruveilhier, and Dr. Budd.[2] It is to elucidate these cases that Brücke's and Bernard's observations before quoted give the most satisfactory clue; and to these must be added the possibility brought forward both by Dr. Budd and Virchow,[3] that in many other diseases there is a tendency to a perverted secretion of the stomach, which probably exercises a peculiarly corrosive influence after death; though, as Virchow[4] has remarked (and his observations are supported by Camerer), the most irritating secretions formed during life appear to have no influence in producing this effect so long as vitality persists.[5]

(*f*) The frequent absence of these softenings has been fully explained by Drs. Budd[6] and Pavy[7] to be due to the alkaline reaction of the blood, neutralizing the acidity of the gastric juice, and also to the proportion borne between the amount of this fluid in the vessels and the acid reaction of the contents of the stomach. Immunity of the posterior wall, while the anterior portion has been affected, has further been explained by the former author to depend on the gravitation of the blood to this part of the stomach; or, as in a case reported by Burns, on the presence of an alkaline dropsical fluid in the cavity of the abdomen.

(*g*) The process of softening appears to be only slightly influenced by external temperature[8]—a fact explicable by the observations of Nasse[9] and Elsaesser,[10] that the cooling of the internal organs of the body is not so rapid during colder seasons as might *à priori* be supposed; and their temperature often remains sufficiently elevated during a period adequate for the production, by self-digestion, of the appearances described.

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[1] See, in support of this view, Virchow, Würzburg Verhandl. i. 296; Archiv Path. Anat. v. 359.

[2] A remarkable instance of this kind is given by Dr. Budd, On the Stomach, 1855, pp. 16, 17.

[3] Würzburg Verhandl. loc. cit.

[4] Archiv, vol. v. p. 360.

[5] It now, however, appears to be probable that this may occasionally occur. See Ulcer, Etiology, also p. 992.

[6] Croonian Lectures, Medical Gazette, 1847; and Diseases of Stomach, 1855.

[7] Phil. Trans. 1863. Treatise on Digestion, 1867.

[8] Elsaesser, loc. cit. 51.

[9] Untersuch. zur Phys. Path. 1835.

[10] Loc. cit. p. 69.

(*h*) Lastly, when food is contained in the stomach, the effects of the fluid chyme on its coats may be influenced both by the amount and by the nature of the food. It has been long known that the gastric juice is only sufficient for the digestion of a certain amount of food,[1] and, therefore, if this be in excess of the powers of the solvent, its action cannot be exerted on the containing organ; while, on the other hand, as pointed out by Dr. Brinton,[2] amylaceous matters, which neutralize less of the gastric juice than albuminoid materials, tend, not only directly by their fermentation (to which allusion has already been made), but also indirectly, by exciting the secretion of an acid fluid, to give rise to this solvent action. Further, as has been stated by Dr. Budd, medicinal substances, and especially alcohol, given shortly before death, may in many cases more or less completely prevent this chemical dissolution.

From the considerations above quoted, it therefore appears that in almost all cases, with the exception of the rare instance before quoted, the changes in the stomach now described must be regarded as the result of post-mortem effects of a purely chemical nature; but that, in a certain number of instances, morbid conditions of the stomach, which tend either to cause an increased secretion of the gastric juice, or to set up fermentative changes in the food (especially when this consists of milk or amylaceous substances), may favour this action. It is, however, one that only very rarely occurs to any appreciable extent until after life has ceased to exist, and the appearances so produced cannot therefore in the majority of cases be regarded as anatomical or pathological conditions to which any of the symptoms occurring during life can, with justice, be attributed.

It seems tolerably certain that these softenings are not identical with those produced by putrefactive changes in other tissues of the body, though these undoubtedly aid in increasing the degree of softness of the membrane. It is not indeed, in all circumstances, easy to discriminate the exact share attributable severally to each of these processes—which must be, to some degree, determined by the amount of cadaveric change observed in other parts. One of the most marked evidences of the latter, though not, comparatively, very frequent, is an emphysematous condition of the coats, arising from the development of gas through putrefaction in the submucous tissue.

It has been already stated that the imbibition of the mucous membrane with hæmatin may occur very early after death, and is due probably to both causes acting conjointly; but the blackened appearance of the blood in the vessels, though partly due to the action of the gases of the gastro-intestinal canal is, in a great measure, the result of the action of the gastric juice, as has also been explained in previous sections.

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[1] For further and exact confirmation of this fact, see Brücke, Sitzber. Wiener Akad. 1859.

[2] Diseases of the Stomach, p. 80.

XIII.—Perforation of the Stomach.

Perforation from within, formerly described as an independent disease, has received no recent trustworthy confirmation. The cases in which it has been described are now almost universally admitted to have resulted from ulcerative processes, or to have arisen from post-mortem softening. The modes of distinguishing these conditions have been already described (see Ulcer). Cases of perforation, due to foreign bodies in its interior, are, however, occasionally met with.[1] An interesting case of perforation of the stomach, caused by an abscess of the liver, which communicated simultaneously with the pericardium, is recorded by Dr. Graves.[2]

XIV.—Rupture of the Stomach.

Rupture has only with the exception of the rarest instances been authentically established in cases of external injury. Ziemssen has, however, seen rupture follow distension through fermentation processes in a stomach which was the seat of stricture arising from the healing of a chronic ulcer.[3] The symptoms have been those of collapse, followed by peritonitis.[4] The occurrence is most likely to happen when the stomach is distended by food at the time of the injury. Whether a fatty degeneration of the muscular coat of the organ may occasion the spontaneous occurrence of this event must remain a subject for further inquiry.

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[1] See a case by Drs. Quain and Bucknill, of perforation of the stomach by a mass of cocoa fibre, swallowed by a lunatic, Path. Soc. Trans. vol. v. Another, of perforation of the duodenum in a lunatic who swallowed spoon-handles and nails, has been recorded by Mr. Poland, quoted by Mr. Pollock, Holmes' Syst. Surg. ii. 470.

[2] Clin. Med. ii. 236.

[3] Sammlung clinischer Vorträge (Volkmann), No. xv. p. 100.

[4] A case of this kind occurring after a fall is recorded by Mr. Moore, Lond. Med. Rev. July 1860. A case is also given by Richerand, Physiol. i. 282, of a woman in whom, after an external injury, a fistula subsequently formed, quoted by Dr. Pollock, Holmes' Surgery, ii.

XV.—TUBERCLE OF THE STOMACH.

This must be regarded as an exceptionally rare disease. Andral[1] says that he only met with it twice. Willigk[2] only found it five times in 1,317 cases. In 141 cases of tubercle in the gastro-intestinal canal, Barthez and Rilliet[3] found the stomach affected in 21. These authors state that its seat is ordinarily in the great curvature, and that in this situation it may sometimes give rise to extensive ulcerations, which may attain the diameter of a five-franc piece.[4]

The author has never seen this disease commencing in the mucous membrane, but he has found a perforation of the stomach caused by tubercles commencing in the peritoneum, passing from without inwards through its coats, and thus following the tendency, observed by Barthez and Rilliet,[5] of tubercles seated on the peritoneal surface of other parts of the canal.

It may possibly be questioned whether the follicular ulcerations of the stomach, so common in phthisis, may not be due to the solitary glands undergoing changes similar to those seen in other portions of the intestine, but the author has not been able to obtain any positive proof of the identity of the process in these parts.

Perforation of the stomach by a tubercular ulcer, commencing in the submucous tissue of the great curvature, and attended by copious hæmatemesis, resulting from the invasion of the gastro-epiploic artery, has been recorded by Bignon.[6] Tubercles existed around this ulceration.

In the majority of cases recorded, no symptoms have been observed. In three only of the cases recorded by Barthez and Rilliet was vomiting present. The disease, in the present state of our knowledge, offers no distinctive features for either diagnosis or treatment.

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[1] Prec. Path. Anat. ii. 178.

[2] Prager Viertel-Jahresch. vol. i. p. 80.

[3] Malad. des Enfants, iii. 183.

[4] See also Weissbach, Canstatt's Jahresb. 1844, iv. 203. Also Valentin, Virchow's Archiv. xliv. p. 306. The latter author distinctly traced tubercular ulceration in the stomach associated with granulations. He questions whether the granulations of tubercle were not secondary to the ulcer of the stomach. Steiner and Neurethe (Prager Viertel-Jahresch. 1865, ii.) have also seen this. I have seen the stomach perforated by tubercle, proceeding apparently from the peritoneum.

[5] Loc. cit. 780.

[6] Gaz. des Hôpitaux, 1853, p. 111.

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